# JOURNAL

## OF THE

## American Veterinary Medical Association

**FORMERLY AMERICAN VETERINARY REVIEW**

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 1230 W. Washington Blvd., Chicago, Ill.





**Vol. LXXX, N. S. Vol. 33** **May, 1932** **No. 5**


## OUR NEW LOCATION

As promised last month, we are now able to give the members of the A. V. M. A. some information about the new location of the official headquarters of the Association, in Chicago.

The new offices are located on the first floor of the Prairie Farmer Building, 1230 West Washington Boulevard, at the corner of Willard Court, about two miles west of the loop district, directly on U. S. 20 and Illinois 5. There is plenty of parking space around the building. We have about 25 per cent more floor space than we had in the Book Building in Detroit, and our rent is about 25 per cent less, the result of getting away from the downtown district. Some part of our saving in rent will be offset by other expenses which we will have to meet in Chicago and which we did not have in our former location, but this was anticipated.

It has been necessary to select and organize an entirely new office staff. In this respect we feel that we have been particularly fortunate in securing the services of three young ladies, whose previous training and experience have been such as to qualify them admirably for their new work. They are rapidly getting oriented, under the able direction of Miss Anderson, whom many members met at the conventions in Lexington, Philadelphia, Minneapolis and Detroit. She came to Chicago and spent a month here helping us to get started.

Radio Station WLS is located in the Prairie Farmer Building, so whenever you hear a program originating in that station, keep in mind that the WLS studio is just two floors above Station AVMA.

Our new telephone number is Haymarket 3254. Give us a ring. Better yet—drop in, the next time you are in the Windy City.

The Prairie Farmer Building

## CAN YOU USE AN ASSISTANT?

Next month our colleges will graduate another crop of veterinarians, the largest since 1923. Last fall there were 267 senior veterinary students registered in the thirteen colleges in the United States and Canada. In all probability about 240 of these will receive their diplomas this year. If these graduates were to be equally distributed throughout the United States and Canada, there would be about four for each state and province. Of course, an even distribution is quite improbable, as the more densely populated localities will absorb more of these new veterinarians than the sparsely settled sections. However, the average of four per state or province is mentioned to show how relatively small the number is. The total for the United States is still considerably below the figure 298, estimated by the A. V. M. A. Committee on Education as the theoretical requirement for maintaining the number of veterinarians at the 1930 level.

Several of the prospective graduates have written us to inquire about positions and locations. Likewise, a number of the students in the lower classes have made similar inquiries, most of these wanting positions with veterinarians during the summer months. This office will attempt to act as a clearing-house for both employer and employe. Veterinarians wanting either graduate or student assistants are invited to communicate with us, stating requirements and giving all possible information in the first letter. The indications are that some of these students seeking positions will be unable to return to college in the fall unless they succeed in securing some form of remunerative employment during the vacation period. Can you use either a student or graduate assistant this summer?

## NOT OUR FAULT

A member recently registered a mild complaint because he had "missed out" on a meeting of a veterinary association that meets regularly twice a year in his territory. This member saw no announcement of the meeting in the JOURNAL and was inclined to blame the editor for failing to announce the meeting. Now, here are the facts:

Over a month before the usual time for the meeting, we wrote a letter to the secretary, asking for information concerning the time and place. The information finally was received, to the effect that the meeting was to be held in February (date given). The letter was received February 2, a week after the February issue of the JOURNAL had gone to press and entirely too late for any announcement of the meeting to be made.

The JOURNAL goes to considerable trouble to secure advance information concerning veterinary meetings, so that this can be published for the benefit of our members. Some veterinary associations that do not have fixed dates for regular meetings apparently do not appreciate the value of fixing dates for meetings as far ahead as possible. This enables the JOURNAL to publish advance announcements, and veterinarians can make their plans to attend to better advantage if they know some time ahead just when the meeting will be held.

We ask the coöperation of secretaries of veterinary associations; state, sectional and local, to this end. Get the information to us not later than the 20th of the month preceding the month in which the meeting is to be held. Earlier, if possible.

## EXECUTIVE BOARD ELECTIONS

The primary elections held in Executive Board districts 1 and 9 came to a close April 23. The canvass of the ballots revealed the candidates listed below as having received the highest number of votes with the following exception: In District 1 (Canada), Dr. George Hilton received a sufficient number of votes to place him among the first five, but a letter received from Dr. Hilton, during the early days of the election, indicated that he preferred not to be a candicate for reëlection to the Executive Board. At this time it would be in order to direct attention to the fact that Dr. Hilton has been a member of the Executive Board since 1918, in which year he succeeded the late Dr. Frederick Torrance. In August, Dr. Hilton will round out a period of fourteen years of continuous service on the Executive Board, a record approached by only one other member of the Association, Dr. T. E. Munce, who served continuously for a period of ten years (1918-1928). During two years (1920-1922) of his service, Dr. Hilton was chairman of the Executive Board.

Ballots containing the names of the five candidates in each district have been mailed to all paid-up members in the two districts. (District 9 comprises New York and the New England States.) The polls will remain open until June 23, which is sixty days before the opening date of the Atlanta convention, as provided in the By-laws.

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Health of Animals Branch, Canada Department of Agriculture. Graduate of Ontario Veterinary College, 1908. Joined A. V. M. A., 1913.

HADWEN, SEYMOUR — Toronto, Ont.
Director of Veterinary Research, Ontario Research Foundation. Graduate of McGill University, 1902. Joined A. V. M. A., 1904. First vice-president, 1928-29.

HALL, ORLAN — Ottawa, Ont.
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1913-15; member of Audit Committee, 1917-18; member of Committee on Resolutions, 1918-21; member of Committee on History, 1923-25; member of Committee on Necrology, 1926-27; member of Committee on Education, 1929-32.

McINTOSH, R. A. Guelph, Ont.

Professor of Obstetrics, Pharmacy and Therapeutics, Ontario Veterinary College. Graduate of McKillip Veterinary College, 1909. Joined A. V. M. A., 1923. Resident secretary for Ontario, 1923-32.

### DISTRICT 9

EICHHORN, ADOLPH Pearl River, N. Y.

Director, Veterinary Department, Lederle Laboratories, Inc., Pearl River, N. Y. Graduate of New York-American Veterinary College, 1900. Joined A. V. M. A., 1901. Vice-president, 1913-16 and 1918-20.

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HARTMAN, PAUL H. 1121-24th Street, N. W., Washington, D. C.
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Vouchers: H. E. Moskey and W. E. Cotton.

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Director of Veterinary Research, Ontario Research Foundation. Graduate of McGill University, 1902. Joined A. V. M. A., 1904. First vice-president, 1928-29.

Hall, Orlan — Ottawa, Ont.
Health of Animals Branch, Canada Department of Agriculture. Graduate of Ontario Veterinary College, 1910. Joined A. V. M. A., 1913. Secretary (1922-1923) and chairman (1923-1925) of Section on Sanitary Science and Police.

McGilvray, C. D. — Guelph, Ont.
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D. V. M., Kansas State College, 1915
Vouchers: C. L. White and A. E. Slocum.

SECORD, ALAN C.
College of Veterinary Medicine, Ohio State University, Columbus, Ohio
B. V. Sc., Ontario Veterinary College, 1929
Vouchers: W. F. Guard and Walter R. Krill.

WOOLFOLK, GEORGE H. 2022 W. Cumberland Ave., Knoxville, Tenn.
D. V. S., Kansas City Veterinary College, 1906
Vouchers: James A. Sluss and M. Jacob.

## Applications Pending

SECOND LISTING

(See April, 1932, JOURNAL)

Aronson, Harry P., 261 W. Sunrise Highway, Freeport, N. Y.
Berroth, Elmo M., 1838 Walker Ave., Kansas City, Kans.
Enge, Edwin C. F., 39th St. & Woodland Ave., Philadelphia, Pa.
Foster, Eugene Z., 312 N. Eutaw St., Baltimore, Md.
Hare, H. Conrad, 4880 Hudson Blvd., West New York, N. J.
Moulthrop, Irwin M., College Park, Md.
Waller, Ernest F., University Farm, Saint Paul, Minn.

The amount which should accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

## COMING VETERINARY MEETINGS

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. May 4, 1932. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

New York City, Veterinary Medical Association of, Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. May 4, 1932. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

Connecticut Veterinary Medical Association. Hotel Elton, Waterbury, Conn. May 4, 1932. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Michigan-Ohio Veterinary Medical Association. Adrian, Mich. May 5, 1932. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. May 10, 1932. Dr. E. E. Sweebe, Secretary, 14th St. and Sheridan Road, North Chicago, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. May 10, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. May 11, 1932. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Hudson Valley Veterinary Medical Society. West Point, N. Y. May 11, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Tulsa County Veterinary Association. Tulsa, Okla. May 12, 1932. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. May 18, 1932. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.

Keystone Veterinary Medical Association. Philadelphia, Pa. May 25, 1932. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Colorado Veterinary Medical Association. Fort Collins, Colo. May 26-27, 1932. Dr. Floyd Cross, Secretary, Fort Collins, Colo.

Southeast Georgia Veterinary Association. Vidalia, Ga. June 1, 1932. Dr. Hugh F. Arundel, Secretary, Box 68, Quitman, Ga.

Oklahoma Veterinary Medical Association. Stillwater, Okla. June 6-7, 1932. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. A. & M. College of Texas, College Station, Texas. June 6-10, 1932. Dr. D. Pearce, Secretary, Box 335, Leonard, Texas.

California State Veterinary Medical Association. Yosemite National Park, Calif. June 20-22, 1932. Dr. W. L. Curtis, Secretary, 1264 W. Second St., Los Angeles, Calif.

Maryland State Veterinary Medical Association. Colonial Hotel, Ocean City, Md. June 23-24,1932. Dr. E. M. Pickens, Secretary, College Park, Md.

Michigan State Veterinary Medical Association. East Lansing, Mich. June 28-29, 1932. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

New York State Veterinary Medical Society. New York State Veterinary College, Ithaca, N. Y. June 29-30, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

North Carolina State Veterinary Medical Association. Joint Meeting with Virginia State Veterinary Medical Association. Henderson, N. C. June 29-30, 1932. Dr. J. Howard Brown, Secretary, Rich Square, N. C.

Virginia State Veterinary Medical Association. (See North Carolina announcement above.) Dr. I. D. Wilson, Secretary, Va. Poly. Inst., Blacksburg, Va.

# THE VALUE OF IODIN IN CATTLE PRACTICE*

*By* E. C. McCulloch, *Madison, Wis.*

*Department of Veterinary Science, University of Wisconsin*

## The Iodin Requirements of Normal Animals

During the thirty-five years since Braumann[1] announced that iodin was a normal constituent of the thyroid gland, it has become well established that iodin is intimately associated with metabolism and that small amounts are required by all animals. Therefore, the prophylactic as well as the therapeutic values of iodin compounds are worthy of attention.

As early as 1907, Marine[2] recognized iodin deficiency, and in reporting upon glandular hyperplasias in the thyroids of dogs, stated that "lack of iodin was the essential deficiency, and iodin, when supplied, quickly overcame needs."

Ten years later, in 1917, Marine and Kimball[3] called attention to the prophylactic value of iodin in the prevention of human goitre. During the same year, Smith[4] pointed out the value of iodin in the prevention of thyroid disturbances in animals, and at this early date wrote:

> If more iodin were fed the pregnant animals in large sections of this continent, especially during the winter months, the young that they produce would be more healthy and more vigorous and the large number of weak and defective young animals that are produced annually would be greatly decreased.

It was at this time that the attention of the veterinary profession was called to the significance of iodin deficiency by Welch,[5] at the Montana Experiment Station, who reported that "iodin supplied to the female breeding stock during gestation is apparently effective as a preventive of goitre in the new-born."

This was followed by the work of Hart and Steenbock,[6] of the Wisconsin Experiment Station, on the hairless-pig malady, in which the role of iodin deficiency was recognized, although the disease was up to that time thought to be associated with rations of high protein levels and low laxative effects.

Kalkus,[7] of the Washington Experiment Station, in 1920, called attention to the fact that in the goitrous districts many apparently normal adult cattle of both sexes were affected with enlarged thyroids, and that the fullness of a cow's neck in the region of the thyroids would naturally tend to hide a slightly en-

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

larged gland. He stated that in the goitrous districts the losses are not confined to the goitrous calves, but that some new-born calves not showing external symptoms of goitre were abnormally weak. At this time he wrote:

> It has been definitely proved that the administration of iodin in various forms and by different methods will prevent goitre and its associated conditions.

Hopkirk and Simpson,[8] working in New Zealand, found that in the iodin-deficient areas the stock was poorly developed, especially the yearling heifers which were small in size; sterility was prevalent and the cream-production was very low, although the pastures appeared to be luxuriant and rich. Upon analysis the soils were found to have the extremely low iodin content of from two to seven parts of iodin in 10,000,000 parts of soil, and produced pasture foliage containing from eleven to fourteen parts of iodin per 100,000,000 parts of dry weight. Milk produced by the cows in this area contained as low as two parts of iodin to 100,000,000 parts of milk.

### Goitre and Hairlessness

In 1924, Forbes and associates[9] recommended that since the deficiency of iodin in feed and water has now been definitely established as the cause of goitre and hairlessness in certain farm animals, the prevalence of these disorders in certain regions unquestionably warrants the administration of iodin.

Clinical experience has taught that goitrous calves are one of the most common and easily recognized results of iodin deficiency, although it is now known that an iodin intake barely sufficient to prevent the extreme symptoms of hairlessness and goitre in the new-born may yet be the cause of serious metabolic disturbances. A decrease in growth and production as well as deficiency of feed utilization may result from an iodin deficiency not severe euough to cause recognizable clinical symptoms. Evvard and Culbertson[10] reported from their experiments, in which traces of potassium iodid were added to the ration of swine, that the feed required for 100 pounds of gain was reduced by iodid feeding in the three experiments, respectively, as follows: 12.5, 9.2 and 8.0 per cent. On the average 10.0 per cent less feed was required with iodid feeding. This was under conditions where there was no clinical evidence of iodin deficiency.

Stiner[11] found that in the recognized iodin-deficient areas the administration of suitable amounts of iodin to milk cows increased the milk yield about eight per cent. Schwaibold and Sharrer[12]

reported an increase in milk yield of over ten per cent following iodin feeding.

On the basis of experimental work carried on at the Rowett Research Institute at Aberdeen, Kelly[13] concluded that the addition of iodin to the ration increased the assimilation and retention of nitrogen and phosphorus, and to a lesser degree that of calcium.

Large areas in the United States, especially along the southeastern boundaries of the Great Lakes and from Lake Michigan northwesterly to the Pacific Coast are known to be deficient in iodin and in such areas this lack of iodin may well be the limiting factor in economical cattle production.

In view of the fact that iodin deficiency has been so forcefully called to our attention and because increases in both growth and production have followed its feeding in iodin-deficient areas, it seems appropriate for veterinarians to give the subject more careful thought and study.

## Sources of Iodin

The principal natural sources of iodin are soil and water. Fortunately when it is lacking, the veterinary practitioner has an inexhaustable supply to draw upon from commercial sources. Accordingly he should determine whether artificial iodization of the animals in his community is needed.

Kalkus suggested a practical and easy method of routine dosage. He recommends that the iodid of potassium be prescribed, as it readily dissolves in water. His preparation consists of one ounce of this salt in a gallon of water. One tablespoon of such a solution will contain approximately two grains, which constitutes the daily dose. Welch[14] expresses the opinion that even one grain of potassium iodid a day is in excess of the amount required. Hart, Steenbock and Morrison[15] suggest that 320 grains (two-thirds of an ounce, troy weight) of the dry potassium or sodium iodid be mixed thoroughly in each 1,000 pounds of the concentrate mixture fed pregnant animals; for stock on pasture the dry potassium iodid may be mixed with the stock salt. In this case it will be necessary to estimate the amount of common salt the animals are consuming daily, and then to mix sufficient of the potassium iodid with the salt so that they will get about two grains a day.

A satisfactory method of giving it to dairy cows is to place tablets containing five grains of potassium iodid in the drinking

cups once or twice a week. These tablets are used for sixty days preceding freshening. Forbes and his associates[9] conclude:

> The use of iodized common salt, which can be purchased in the market, meets all requirements.

### Danger of Over-Dosage

Overdoses of iodin in any form have poisonous effects, which are first shown by watering of the eyes, slobbering at the mouth and running at the nose.

Indiscriminate dosage with iodin is to be avoided. Hart and Steenbock,[6] as early as 1918, warned:

> We are of the opinion, for the present at least, that we have not reached the stage where it is wise to advocate the *general* use of iodin in the feed of all brood sows.

Evvard, Lamb and Gaessler[16] warn against excessive dosage with iodin. They found that experiment ewes, fed from 2.02 to 6.09 grains of potassium iodid daily during the period of gestation, were not able to consume as much feed as the control animals, and that while the ewes appeared to be unaffected, the mortality of their young was from 43 per cent to 92 per cent greater than in the control lots. Later experiments by the same workers demonstrated that a dosage of from one-half to one grain of potassium iodid per week was sufficient to protect ewes against the symptoms of iodin deficiency.

That excessive iodization might be injurious is indicated by the work of Brande and Schwarzman,[17] who found that the administration of excessive amounts of iodin to sexually mature female rabbits produced destructive changes in the ovaries, manifested by degeneration and a disappearance of the follicle apparatus.

The indirect iodization of milk by feeding iodids to the milking cows has been suggested, and might lead to injurious overdoses with iodin. At the Ohio Experiment Station[18] it was found that cows fed potassium iodid produced milk varying in iodin content from 0.3 to 0.7 mg. per quart and on this basis from 4.0 to 8.0 per cent of the iodin fed appeared in the milk. The detectable iodin in the milk of two cows thus fed for one and one-half years disappeared in six days after feeding was discontinued, and no toxic effects of the continued iodization were noticed.

### Therapeutic Uses of Iodin

The use of iodin internally, as recommended in 1865 by Thomassen of Utrecht,[19] on the basis of results obtained in eighty cases

of *Actinomyces bovis* infection, is now universally accepted as the classical method for the treatment of both animal and human patients suffering from infection with this microörganism.

Kingman[20] recommends, in addition to opening and evacuating the actinomycotic abscess, the administration by the mouth of appropriate doses of potassium iodid, using sixty to ninety grains a day for a yearling calf and three drams a day for a thousand-pound cow and continued for five days. If the resolution of the tumor is not complete in from ten to fourteen days, he recommends that the treatment be repeated.

Lytle[21] describes an interesting operation in which, after suitable restraint and anesthesia, five or six ounces of Lugol's solution or tincture of iodin is injected into the tissues invaded by *Actinomyces bovis*, attempting to distribute the iodin solution as thoroughly as possible throughout the whole honeycombed, bony structure. This surgical treatment is supplemented with intravenous injections of sodium iodid, using approximately half-ounce doses dissolved in 40 to 60 cc of sterile water. Excellent clinical results have followed this mode of treatment, and it appears to be well adapted to those numerous cases in which the practitioner must obtain results from a single treatment.

Tincture of iodin has yielded good results in the treatment of foot-rot, and appears to be especially efficient in the destruction of all forms of *Actinomyces necrophorus* infection.

Ringworm of cattle readily yields to local iodin therapy. Exceptionally encouraging results have been secured from the local application of iodin in alcohol, two ounces of iodin being dissolved in one gallon of commercially denatured alcohol. This is much less expensive than the official tincture and for this purpose appears to be equally effective. Because of the irritating compounds which result from the oxidation of methyl alcohol, the alcohol used should not contain this denaturant.

Potassium iodid forms soluble double salts with the metals and therefore is useful as an antidote in chronic lead, arsenic, mercury and zinc poisoning.

### Iodin as an Alterative

At the present time most investigators believe that the only important physiological effect of iodin is its influence upon metabolism. This action is not entirely understood, although we now know that the continued use of this drug increases the rapidity of nitrogenous breakdown. When taken into the stomach,

iodin preparations are readily absorbed and circulate in the blood-stream chiefly in the form of an iodid. Within the last few months, Tunnicliff[22] has found that animals receiving intravenous injections of dilute sodium iodid solutions showed a marked increase of phagocytosis in the blood. This may be the foundation of the widely recognized but little understood value of the iodin compounds.

### Iodin as a Disinfectant

Although Coindet,[23] of Geneva, in 1819, employed iodin as a medicine, the earliest reference to its use as a disinfectant appears to be in the fourth edition of Bryant's[24] "Practice of Surgery," published in 1884, in which he wrote:

> Those who disregard atmospheric germs and yet highly value means for purifying wound surfaces, will use antiseptic irrigation of the wound with a lotion of iodin, made by adding ten drops of liquor iodi to the ounce of water. I have employed iodin lotion for years and prefer it to any other.

It was not, however, until the first decade of this century that iodin began to receive general recognition as a most useful skin disinfectant.

Woodbury,[25] as late as 1912, complained of the lack of agreement concerning preoperative skin disinfection and called attention to the excellence of tincture of iodin for this purpose. Since that time tincture of iodin has served as *the* standard skin disinfectant with which other disinfectants have been compared.

During the last decade several newly introduced chemical compounds have been widely advertised and heralded by research workers as well as by their manufacturers as being distinctly superior to the older iodin compounds. In practically all instances their claims have been supported by critical data based upon laboratory results. It will be the purpose of this part of this paper to analyze in part these claims and results and in so far as possible to adapt them to the conditions which the practitioner meets.

It is unfortunate that in the testing of the germicidal value of a substance, apparently insignificant variations in laboratory technic may greatly alter the experimental results obtained. This is particularly true when the germicidal value of substances of markedly different chemical nature are being compared. In many instances the newer synthetic chemical compounds possess an inhibitory value far in excess of their disinfectant action and in at least some of the recorded laboratory tests it is

probable that sufficient chemical has been carried over into the culture tubes to inhibit the growth of the microörganisms, with the result that the substance under test has received undeserved credit.

This appears to be particularly true in the study of agents employed for skin disinfection. The correct evaluation of disinfectants when applied to the skin is made even more difficult by the self-disinfecting power possessed by living cutaneous tissue. Recently, investigators[26] have reported that the intact outermost layer of skin possesses sufficient disinfecting powers to destroy cultures of certain organisms within thirty minutes. Again, some chemical agents are powerful destroyers of germ life when acting in a medium of distilled water; but become almost inert in the presence of discharges, blood-serum and other albumin-containing products. Still other agents are extremely selective in their action and are effective against only one group of organisms, exerting practically no effect upon other pathogenic microbial life. Manufacturers of germicides have not been aware of this situation and in presenting their product in the most favorable light have often confused the men in the field by their claims. Research workers using different technics have published results that varied widely, thus adding to the confusion.

## The Ideal Disinfectant

Obviously, the ideal disinfectant would be one that would kill the infecting agent without at the same time causing injury to the tissue cells. Using cultures of living tissue from the spleen and staphylococci from the throat and from a case of furunculosis, Lambert,[27] in 1916, compared the disinfecting ability of the tissue toxicity of numerous compounds. He concluded that "iodin stands out as the one chemical tested to which cells were found to be more resistant than were staphylococci."

Scott and Hill[28] called attention to the fact that while aqueous solutions of mercurochrome are bactericidal when tested in pure culture, yet such solutions were uniformly less efficient than iodin solutions in the actual disinfection of skin surfaces.

Reddish,[29] after comparing the action of tincture of iodin and a 5 per cent alcoholic solution of mercurochrome on *Cl. tetani* and *Cl. welchii*, found that neither organism was killed after exposure for two hours to the mercurochrome solution, while the tincture of iodin killed *Cl. tetani* in one hour and *Cl. welchii* in thirty minutes.

Simmons[30] observed that a 5 per cent solution of mercurochrome in 50 per cent alcohol compared rather favorably with tincture of iodin, in its action on some of the normal throat and skin organisms when used on skin surfaces, but that it was less efficient in destroying certain spore-forming bacteria. In his experiment the abdominal skin of each of a series of white mice was contaminated with a ten-day-old culture of *B. anthracis* which contained many spores. When dry the contaminated skin was liberally painted with the solution to be tested, and later a small piece of the treated skin was excised, inverted and inserted into the wound. All of the mercurochrome-treated animals died of anthrax, while all of the iodin-treated animals survived. One might criticize Simmons' conclusions in this experiment on the grounds that only nine animals were used and the results might have been due partially to chance. Simmons reported on experimental work in which broth cultures of *Staph. aureus*, *Str. pyogenes*, *Str. scarletinae*, *E. coli* and *Cl. welchii* were rubbed over the dried shaven skin of rabbits. After the contaminated skin had dried for one hour it was treated with one of the various disinfectant solutions under study, and both surface and deep skin scrapings were made. Under these conditions, tincture of iodin gave results much superior to those obtained from the use of either the aqueous or alcoholic solutions of mercurochrome.

### Action of Mercurochrome

Rodriguez,[31] in testing the value of various agents in sterilizing areas of the mucosa of the human mouth, found a 2 per cent aqueous solution of mercurochrome to be too feeble to be depended upon as a surface disinfectant on the oral mucous membrane. A 5 per cent mercurochrome solution in alcohol, or in an alcohol-acetone mixture, possessed decided advantages over the aqueous solution, but failed in too large a proportion of the cases to be considered effective in surface disinfection of the oral mucous membranes. In contrast he found 3.5 per cent of even 1.75 per cent of iodin in glycerin to be efficient in destruction of organisms on the oral mucosa. The full-strength tincture of iodin is now seldom used on mucous membranes. In such strengths it acts as a powerful irritant, and in susceptible individuals may produce local effects of a serious nature. Even the 3.5 per cent alcoholic solution, although possessing an effective bactericidal action, may not be well tolerated by the mucosa. Rodriguez found that the combination of iodin with

glycerin; that is, glycerin being used as the diluent of the standard tincture, instead of alcohol, resulted in a preparation easily tolerated and devoid of any irritating effects, while maintaining the desired germicidal properties.

In contrast, Reddish and Drake,[32] concluded from their experiments that 2 per cent mercurochrome in aqueous alcohol-acetone solution was as efficient as tincture of iodin in disinfecting the unbroken skin.

Scott, Hill and Ellis[28] also conclude from their experimental work that 2 per cent mercurochrome in an aqueous alcohol-acetone solution or tincture of iodin are equally effective in the sterilization of the unbroken skin, while the 2 per cent aqueous solution of mercurochrome is not efficient for this purpose.

Birkhaug[33] compared the germicidal efficiency of metaphen, hexylresorcinol, mercuric chlorid, mercurochrome and tincture of iodin against *Staph. aureus*, *Str. hemolyticus*, *E. coli*, *B. anthracis*, and *B. subtilis*. Standard conditions were maintained throughout the test and the dilution was determined that would kill in ten but not in five minutes. Tincture of iodin killed *Staph. aureus* in a dilution of 1-3000, *Str. hemolyticus* in 1-4000, *E. coli* in 1-3000 and *B. anthracis* and *B. subtilis* in 1-4000. Metaphen killed *Staph. aureus* in a dilution of 1-120,000, *Str. hemolyticus* in 1-90,000, *E. coli* in 1-20,000, and *B. anthracis* and *B. subtilis* in 1-40,000. At first glance this might appear to indicate a decided advantage of metaphen over the official tincture of iodin. However, these data are based upon the actual metaphen content of the solution, while commercially, metaphen is marketed in dilutions of 1-500 and 1-2500. Comparing the 1-500 solution of metaphen with tincture of iodin, we find the disinfectant dilution of the commercial metaphen solution to be 1-240 against *Staph. aureus*, 1-180 against *Str. hemolyticus*, 1-40 against *E. coli* and 1-80 against *B. anthracis* and *B. subtilis*. Under the same conditions, tincture of iodin killed these organisms in dilutions of 1-3000 to 1-4000. Metaphen, even in the 1-500 dilution, is more expensive than tincture of iodin,thus making disinfection approximately twenty-five times as costly as when tincture of iodin is used.

### Hexylresorcinol Recently Introduced

Hexylresorcinol is another recently developed disinfectant that is commonly recommended as a substitute for tincture of iodin. As a 1-1000 aqueous glycerin solution, it is sold commercially

as "S. T. 37." Data based upon the pure chemical in the solution indicated that dilutions of from 1-4000 to 1-16,000 were necessary to destroy the organisms under test, which means that on the basis of germicidal efficiency, 1-4 to 1-16 dilutions of the commercial product must be compared with the 1-3000 to 1-4000 dilutions of tincture of iodin which destroyed the same organisms under the same conditions. This gives a differentiation of cost of approximately 250 times in favor of tincture of iodin over commercial preparations of hexylresorcinol. Leonard[34] maintains that hexlyresorcinol solutions are not adapted to skin sterilization, being devoid of any fat dissolving power and lacking ability to penetrate below the most superficial layer of the skin.

As the germicidal action of iodin is practically the same against all types of microörganisms and as the practitioner has little opportunity to determine the type of organism to be destroyed, it behooves him to use iodin or other agents that are capable of destroying any and all pathogenic microörganisms.

### Preoperative Preparation of the Skin

The skin of cattle is rich in microörganisms, not only on its surface, but also in its thickness, especially in the epidermis and the ducts of the glands. Because moisture causes swelling of the epithelial cells, leading to obstruction of the intercellular spaces and the hair follicles, dry shaving is to be recommended. This allows the tincture of iodin to penetrate and come into direct contact with the microörganisms.

D. Curri,[35] after using iodin in skin disinfection with invariably good results for 20,000 operations extending over twenty-two years, writes:

> The "urge" to discover newer and possibly better methods has led to failure to appreciate this method at its true value.

### Iodin Ointments

In contrast to the efficiency of the alcoholic or alcohol-glycerin solutions of iodin, oily iodin preparations do not appear to be able to exert a marked disinfectant action on the surrounding tissues. When applied directly to the surface of an agar culture of *Staph. aureus*, iodin ointments failed to inhibit the growth of the organism. Archibald and Brown[36] report an experiment in which equal quantities of sputum and iodized vegetable oil which contained forty per cent iodin were mixed and held at room temperature for eighteen days. At this time it was found

that the sputum was decomposed and when inoculated on plates, pseudodiphtheriods, short-chain streptococci and staphylococci were found. Whatever therapeutic value is obtained from the use of iodin ointments does not appear to be the result of direct bactericidal action.

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# FIELD AND EXPERIMENTAL STUDIES OF SHEEP DISEASES*

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In the East, where our flocks are small and have more or less individual care, our disease problems are usually limited to the more commonly known diseases, and those of parasitic origin. The virulent transmissible diseases do not seem to be so prevalent.

There is no mystery about the care, handling and management of sheep. The veterinarian who will apply his knowledge of veterinary medicine and animal husbandry can become proficient in sheep practice.

Sheep men as well as other stockmen, have certain terms and expressions, and to be successful among them one should be acquainted with the vernacular and customs, as, for instance, unimportant as it may seem, you will be judged as to your ability by the way you catch and handle a sheep.

The usual way, when they are flocked together, is to grasp one by the hind leg, above the hock and quickly slip one arm around the breast, the other one around the rump. Never grasp a sheep by the wool.

"Flushing" is a term used in connection with the breeding of ewes, meaning increased feeding prior to breeding in order to increase, if possible, the lamb crop. "Tagging" is another term, meaning clipping the wool around the dock and hind part before breeding, also around the udder prior to lambing. These are a few of the most-used terms.

Sheep are ruminants and are subject to many of the same diseases as the larger ruminants, so the experience acquired in diagnosing and treating diseases of ruminants can be applied in a general way to sheep.

*Dentition:* A sheep's mouth is similar to that of other ruminants. They have 32 teeth: 8 incisors and 24 molars. Lambs have a full set of incisors when they are 30 days old. They are replaced by permanent teeth. The two central permanent incisors appear when the lambs are 12 to 15 months old, the first intermediates at 18 to 24 months, the second intermediates at 2½ to 3 years, and the corner teeth just before the sheep is

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4 years old. After this time, age is determined by wear. Old sheep show what is termed a "broken mouth."

*Breeding:* Estrum begins in lambs when about 6 months old, but it is not advisable to let them breed until they are 15 to 18 months old. In most breeds the period of estrum, in aged ewes, comes 6 and 7 months after lambing, but the Dorsets or Merinos may come in heat much sooner. In some cases Dorsets have been known to come in heat a week or ten days after lambing. The period of estrum is 1 to 3 days and recurs in from 13 to 19 days. The period of gestation averages about 150 days, or approximately five months.

*Physical examination:* The physical examination of sheep should be made along the same general lines as those used when examining the larger ruminants. Examine the visible mucous membranes, take the temperature, which is normally about 103° F. and note the condition of the feces and the general appearance of the animal. The pulse and respiration are not dependable. Observe the condition and color of the skin by parting the wool on the sides of the body. Examine all parts of the body carefully for wounds. This is particularly necessary in warm months, as concealed wounds often become infested with maggots. A thorough examination of the feet must be made for cuts, wounds or foot-rot, and a sample of the feces should be obtained for laboratory examination for parasite eggs.

## Medication

Administering medicine is a simple matter when once understood. To administer liquids, the sheep should be backed into a corner, standing on all four feet. The operator should stand astride the sheep's neck, gripping it firmly with his knees; place his first and second fingers in the interdental space in the mouth, with thumb on the nose, in this way holding the mouth open, so that the drench may be administered slowly with a dose-syringe, or a small mouthed bottle. During this operation never permit the nose of the sheep to be elevated above the level of the eyes. This is important in order to prevent the drench from entering the lungs.

Administering medicine by use of the stomach-tube is safe and practical when once understood. For this purpose the regular hog stomach-tube outfit is used, consisting of a curved metal pipe and rubber tube. The rubber tube can be either pressure hose or rather stiff rubber. Before passing the rubber tube, the

metal pipe is placed in the mouth, as far back as possible on the tongue, with the curved end downward. It is then easy to pass the rubber tube through it. The only difficulties and fatalities that we have ever experienced were when the metal tube had not been inserted far enough in the mouth. In this case, when the rubber tube was passed, it entered the trachea.

*Capsuling:* It is not so easy to capsule sheep as one might think. Unless the capsule is carried well back on the tongue, the sheep will spit it out very readily. For this purpose we use the water capsule-gun, which has a cylinder and the tube slightly curved, attached to the ordinary two-ounce dose-syringe.

### Diseases of the Digestive Tract

The common diseases usually met with in sheep are those of the digestive tract and are due most frequently to faulty flock management and can usually be overcome by correcting the diet and treating the individuals as symptoms are presented. Some of these digestive disturbances are bloating, impaction of the rumen, constipation and diarrhea. These conditions should be treated along the same general lines that cattle, affected in the same manner, would be treated, remembering that the doseage for sheep is about one-tenth to one-fifth of the dose for the average cow, depending on the weight of the sheep.

### Diseases of the Reproductive System

We find many of the diseases of the reproductive system that are found in cows. Similar symptoms are presented and they should be treated in the same general way.

*Acidosis:* This condition, also known as pregnancy disease, seems to be more prevalent in recent years. It affects the ewe a short time prior to lambing. The symptoms, as described by Dimock, are: "lagging behind, standing alone, loss of appetite, nervousness, irritability, grinding the teeth, walking in circles, standing with head pressed against a hard object and partial or complete blindness. Later the animal is unable to stand, has partial or complete coma with convulsions or spasms when disturbed." This is said to be a nutritional disease due to deficiency of calcium. Duration of the disease is 6 to 7 days. The losses in some flocks may run as high as 15 to 20 per cent.

Treatment is not satisfactory. Cod-liver oil or phosphate of lime may be used. Purgatives are indicated, such as Epsom salts, and succulent food should be provided. Preventive measures should be used on the unaffected ewes in the flock, in

the form of enforced exercise and a properly balanced grain ration with alfalfa or clover hay. This ration has a high calcium content. In addition to this ration, a mixture of 1 part fine ground limestone, 1 part of steamed bone meal and 3 parts of salt should be used in place of salt alone. The use of calcium lactate has been suggested as a preventive, and calcium gluconate for affected animals.

### Necrobacillosis

The bacillus of necrosis is responsible for several diseases of sheep and lambs, namely: sore mouth of lambs, navel infection of young lambs, lip-and-leg ulceration, so-called venereal disease and foot-rot. All of these are due primarily to infection with the necrosis bacillus, and one of them may be present without any of the others developing. These diseases are transmitted by the infective material from a diseased sheep being carried into a wound or abrasion on a healthy sheep. Each one of these diseases presents different symptoms.

*Sore mouth in lambs:* Symptoms include water blisters on the upper or lower lip, or near the nostrils. These later develop into ulcers.

*Navel-cord imfection:* The organism enters through the unhealed cord soon after birth. There is no external evidence of disease, but the lamb seems dull and weak. This generally occurs before it is ten days old. On postmortem the liver shows small, characteristic abscesses, containing numerous necrosis bacilli.

*Lip-and-leg ulceration:* Generally affects the full-grown sheep, and the characteristic ulceration appears on the lips and on the legs about the ankles.

*Foot-rot:* The sheep are lame, often with swellings and suppuration between the claws. The foot is hot and the claws spread apart, due to the swelling, frequently with abscess formation. The foot-rot described is the infectious form, and is very difficult to control, as it requires persistent, careful treatment, and in many flocks this is not carried out satisfactorily. There is a simpler form of foot-rot frequently called foot-scald. This is due to wet pastures and insanitary conditions, and responds readily to treatment after the cause has been removed.

The general treatment for the various forms of necrobacillosis is the external use of tincture of iodin, or a solution of dilute nitric acid, or a 5 per cent solution of acetic acid (or common

vinegar). In using nitric acid solution, care must be taken to prevent the acid from reaching any healthy tissue. Tincture of iodin is preferable in the navel-cord infection of lambs. Many cases of foot-rot require considerable surgery before the application of medicinal treatment. Recently the use of tartar emetic incorporated in lanolin has been recommended.

### Hemorrhagic Septicemia

The cause of this disease is the *Pasteurella oviseptica*. Often this organism is found in the air passages of healthy sheep and remains latent unless the normal condition of the animal is lowered by sudden changes of weather, or by exhaustion from shipping, or any cause that may lower its resistance to infection.

*Symptoms:* These vary in different outbreaks, which seems to be characteristic of this disease in all classes of animals. There is an acute and a chronic form. The acute form runs a course of two to five days, with loss of appetite, elevation of temperature, standing with head low, muscles trembling, weakness and difficult breathing. Pneumonia may be present, often colicy pains, with diarrhea streaked with blood, and bad odor. The chronic form may last from one to three weeks. The symptoms are less severe, showing cough, rapid respiration, a discharge from the nose, all indicating lung affection. The losses are heavy in the acute form in lambs. The older sheep generally show the chronic form, with much less loss.

*Postmortem:* The mucous membrane covering any of the organs, or the pleura, may show congestion, or hemorrhagic spots; lesions of pneumonia are frequently present, and a serous or fibrinous exudate is frequently found in the thoracic and abdominal cavities.

*Diagnosis:* This should be confirmed by laboratory examination, with the isolation of the organism.

*Treatment:* Curative treatment is not very satisfactory but preventive measures consist in isolating the affected animals, cleaning and disinfecting the stables, and the use of biologics.

### Diseases of Lambs

*Joint-ill:* Also called pyemic arthritis, is caused by navel-cord infection. Treatment is unsatisfactory; prevention is better. The ewe should lamb in a clean place, and the navel-cord of the lamb should be painted with iodin as soon as possible after birth.

*Goitre*: In some sections of the country this is a common condition in young lambs. It can be controlled to a great extent

through the pregnant ewe. During the latter half of the period of pregnancy she should be given iodide of potash in 1- to 2-gr. doses daily. The use of iodized salt is highly recommended during the entire winter. Iodized salt is prepared by adding 1 pound of granular potassium iodide (powdered) to 300 pounds of salt. Mix thoroughly and feed as ordinary salt.

*Lame, stiff, stilty lambs:* A condition often confused with joint-ill, but in this disease the infection does not occur so early in life. The lambs affected are usually from four to eight weeks old and first show symptoms of stiffness in their legs and walk with a stilty gait. As the disease progresses it becomes difficult for them to rise after lying down, but once on their feet they are able to walk. The hind quarters are usually affected. The appetite is not much impaired, but death may occur from starvation as the lamb cannot walk about to get its food. The cause of this disease is unknown and the treatment unsatisfactory. Cod-liver oil may be used in 2-dram doses. Postmortem presents whitish areas in the affected muscles. Bacteriological findings are negative. Some think it is a mineral deficiency.

*Scours:* When seen in nursing lambs, scours is generally due to some sudden change in the diet of the ewes, such as from dry to succulent food, which changes the character of the milk. Exposure and chilling may be another cause. Give a small dose (2 drams) of castor oil in milk, followed by some intestinal antiseptic, such as sulpho-carbolates.

*Sore eyes:* This trouble is prevalent in many flocks. The true cause is not known, but it is thought to be associated with insanitary conditions of the lambing-pens, and the filthy condition of the ewe. As a preventive, provide clean, well-bedded quarters. Clip the dirty wool from around the dock and udder of the ewe, so the face of the new-born lamb will not come in contact with filth when it nurses. Nitrate of silver solution (2 grains to the ounce of distilled water) gives best results. Prognosis is not always favorable, as blindness may result.

*Turned-in eyelids:* This condition often occurs and can be corrected by a single suture through the lid, to the skin above, just enough to hold it out for a few days.

### Coccidiosis

This disease is becoming more common of late years. It is caused by a protozoan parasite, the *Eimeria faurei.* This disease affects the young particularly.

Symptoms are a foul-smelling diarrhea, which is usually bloody; emaciation, weakness, loss of appetite. Diagnosis is difficult and should be confirmed by laboratory examination.

*Postmortem:* We may find enteritis, and a thickening of the wall of the small intestine; also small whitish areas may be noticed in this organ, varying in size from a pin-point to a pea.

Treatment is not satisfactory, but sulpho-carbolates may be used or salol, or tannic acid in quarter- to half-dram doses.

Recently, at the Bureau's experiment farm, we found lambs, a month old, heavily infested with coccidia. Just how much damage coccidia are doing to lambs and sheep, we are not prepared to say.

## Postmortem Technic

The method of conducting a postmortem examination of sheep is like that of any other animal, but care must be used to detect the presence of parasites. First, examine all parts of the skin for external parasites, wounds and maggots. Examine the lips, mouth and feet for diseases due to the necrobacillosis organism, and the nose and sinuses for grub. After this the carcass should be opened and an examination of the heart and lungs made for any pathological changes. The trachea and bronchi should be opened carefully in order to detect the presence of lung worms. The fourth stomach must be examined for any pathological condition, and close observation must be made of all mucous folds for stomach worms. The intestinal tract, liver, kidneys and bladder should be examined for any abnormal condition, and the entire intestinal tract slit open so that it may be examined for worms and nodules.

## Parasitic Diseases

The extent of parasitic invasion in sheep in this country is not fully realized by many sheep men and veterinarians. The losses due to parasites are enormous and ways and means for their control must be worked out and put in operation.

This is a problem for the veterinarian, and sheep-owners will look to them for the solution. The parasitic diseases are by far the most important of all sheep diseases. Sheep seem to be particularly susceptible to parasitic invasion. It has been estimated that 70 to 80 per cent of all diseases affecting sheep are due to parasites. The internal parasitic diseases are more common than the external, and generally more serious.

I will not detail these parasitic diseases, but wish to mention that the external parasites that are the most serious are lice, ticks and scab mites.

Treatment for all these external parasites is the dipping of all animals in the flock. The dipping material used for ticks and lice is generally some one of the coal-tar products. For scab mites use lime and sulphur, or nicotin dip.

*Internal parasites:* Rather than attempt to discuss all of the internal parasites of sheep, I will call attention to those which are considered the most destructive to the flocks of the eastern and central states.

The most common are the large stomach worm (*Haemonchus contortus*), the small stomach worm (*Ostertagia circumcincta*), the nodular worm (*Oesophagostomum columbianum*), the lung worm (*Dictyocaulus filaria*), the whip worm (*Trichuris ovis*), the hookworm (*Bunostromum trigonocephalum*), and the tapeworm (*Moniezia expansa*). In addition there is grub in the head, caused by the larval form of the *Oestrus ovis*.

In sheep the greatest losses are caused by stomach worms, nodular worms and lung worms. Lambs are more frequently infested with stomach worms and tapeworms

The general symptoms of the presence of internal parasites are unthriftiness, emaciation, paleness of the visible mucous membranes and skin, dullness, frequently diarrhea, but no elevation of temperature. A sample of the feces should be collected for laboratory examination, to determine the presence of parasite ova.

*Manner of infestation:* The eggs or larvae are deposited with the feces and undergo further development. After reaching the infective stage they become attached to grasses, hay, straw and other materials which may be ingested by the sheep to complete the cycle.

The control of internal parasites is a many-sided problem. For several years the Pennsylvania Bureau of Animal Industry has been carrying on some work with sheep to ascertain if it is possible, by use of anthelmintic drugs administered at three-week intervals, to keep them practically free of parasites.

In 1925, we purchased 48 infested ewes and have maintained this flock at our laboratory farm for study and observation. At the time of purchase, a sample of the feces of each ewe was examined for parasite eggs, and all were found to be heavily infested. Nothing was done to control this condition, but sub-

sequent examinations of fecal samples at intervals indicated that the sheep were still heavily infested. In 1928, we outlined the following plan of treatment for the control of parasites in this flock:

Every three weeks all animals were weighed and feces taken for laboratory examination. They were drenched with 3 ounces of a 1½ per cent solution of copper sulfate, to which had been added 24 minims of Blackleaf 40. These treatments were continued at regular intervals until all the animals had been slaughtered. The majority of them were killed in April, May and June, 1931. Careful postmortem examinations were made and worm-counts recorded. The average weight of these ewes in October, 1928, when this treatment was started, was approximately 82 pounds. In October, 1930, their weight was a fraction over 88 pounds, an increase of a little more than 6 pounds per ewe.

TABLE I—*Postmortem findings. (Each sheep received 3 ounces of 1½ per cent solution of copper sulfate containing 24 minims of Blackleaf 40 every 3 weeks.)*

| Sheep | Parasites Found on Postmortem | | | | | | | | Slaughtered | Strongyloid Egg-Count Postmortem |
|---|---|---|---|---|---|---|---|---|---|---|
| | Ostertagia Circumcincta | Haemonchus Contortus | Nematodirus Filicollis | Oesophagostomum Columbianum | Bunostromum Trigonocephalum | Trichuris Ovis | Cooperia Curticei | Monizia Expansa | | |
| 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 7–29 | 0 |
| 2 | 1 | 0 | 0 | 0 | 3 | 1 | 0 | 0 | 6–31 | 4 |
| 4 | 69 | 0 | 2 | 4 | 0 | 0 | 0 | 0 | 4–31 | 10 |
| 7 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10–29 | 0 |
| 9 | 25 | 0 | 2 | 6 | 1 | 0 | 0 | 0 | 5–31 | 0 |
| 10 | 4 | 25 | 0 | 5 | 9 | 0 | 0 | 0 | 5–31 | 9 |
| 13 | 54 | 233 | 0 | 1 | 0 | 0 | 0 | 0 | 5–31 | 77 |
| 14 | 0 | 12 | 0 | 0 | 0 | 0 | 0 | 0 | 6–30 | 5 |
| 15 | 9 | 0 | 0 | 7 | 0 | 0 | 0 | 0 | 6–31 | 0 |
| 16 | 2 | 4 | 0 | 11 | 0 | 2 | 0 | 0 | 5–31 | 17 |
| 17 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6–31 | 4 |
| 19 | 40 | 151 | 82 | 8 | 0 | 0 | 0 | 0 | 4–31 | 267 |
| 20 | 2 | 7 | 0 | 4 | 1 | 0 | 16 | 0 | 5–31 | 1 |
| 21 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 12–30 | 8 |
| 22 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 5–29 | 0 |
| 26 | 22 | 3 | 0 | 4 | 0 | 0 | 0 | 0 | 4–31 | 7 |
| 31 | 0 | 3 | 0 | 4 | 7 | 0 | 0 | 0 | 5–31 | 7 |
| 37 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6–31 | 0 |
| 39 | 0 | 1 | 0 | 3 | 0 | 0 | 0 | 0 | 6–31 | 0 |
| 42 | 2 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 5–31 | 2 |
| 43 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6–30 | 0 |
| 46 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 6–31 | 0 |
| 30 | 284 | 64 | 5 | 7 | 0 | 0 | 82 | 1 | 7–31 | 150 |

## Data

The accompanying tables show the number of parasites found on postmortem, as well as the number of eggs found on fecal examination at the time of slaughter.

In table I are shown the animals in the original, or infected, flock. At the time treatment was started, in January, 1928, all were heavily infested. When slaughtered, five of the twenty-three animals showed no parasites, and others were but slightly infested. The animals with the largest number of parasites also showed the highest egg-counts on fecal examination. It appears that the treatment outlined controlled the infestation, as determined by postmortem and as indicated by table I.

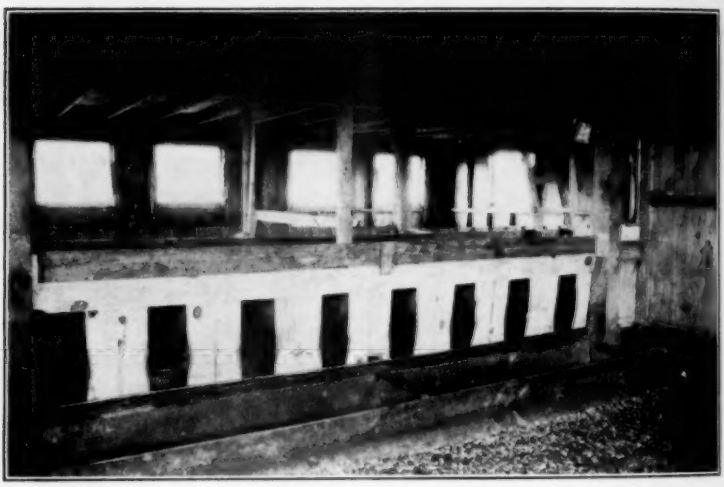

Fig. 1. Main sheep barn, showing original hay-feeding racks, from rear.

In 1928, to prevent infestation in the lambs born from these infested ewes, we attempted to work out a plan of flock management that would prevent the food of these lambs from being contaminated by the infested feces of the ewes.

In order to do this, a practical, inexpensive hay-rack was constructed. The hay-rack is set a foot from the floor and is constructed of slats, wide enough apart to admit a sheep's head. About 18 inches back of the rack a 10-inch board is set on edge. To reach the hay, sheep must step over this board with the front feet, and, to keep them from getting in with all four feet, strips are nailed every 18 inches from the board to the rack. This

simple device prevents them from dragging the hay out and tramping it under foot.

The lambs raised under this system in 1928 were stabled with the ewes in a shed having this specially designed hay-rack and lamb-creep. The ewes and lambs were run together until June 7, when the lambs were weaned. The majority of the lambs were 3 months old, but a few had reached 6 months of age. Regular examinations of the feces had been made and the weights recorded every 3 weeks. At the time they were turned to pasture, all lambs were negative, as indicated by absence of ova from the feces.

The samples of feces examined July 12 and July 26 showed 8 of the lambs to be slightly infested. Treatment was started at this time, each animal receiving 2.5 cc of tetrachlorethylene in a capsule. This treatment was continued at three-week intervals until April 9, 1929. The feces report at this date indicated that all lambs except three (8, 39 and 47) were negative.

TABLE II—*Postmortem findings.*

| | GROUP 1 | | | | | GROUP 2 | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Treatment | 5 cc tetrachlorethylene every 3 weeks | | | | | 3 oz. 1½% copper sulfate solution and 24 min. Blackleaf 40 every 3 weeks | | | | | |
| Sheep | 4 | 13 | 31 | 37 | 46 | 9 | 11 | 21 | 30 | 33 | 43 |
| Parasite | Number found postmortem | | | | | | | | | | |
| *Ostertagia circumcincta* | 0 | 1 | 0 | 2 | 0 | 0 | 9 | 35 | 0 | 0 | 0 |
| *Haemonchus contortus* | 9 | 0 | 0 | 0 | 0 | 1 | 0 | 8 | 6 | 2 | 2 |
| *Nematodirus filicollis* | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 8 | 0 | 0 | 0 |
| *Oesophagostomum columbianum* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Bunostromum trigonocephalum* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Trichuris ovis* | 0 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 0 |
| *Cooperia curticei* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| *Trichostrongylus extenuatus* | 0 | 0 | 0 | 0 | 1 | 0 | 5 | 7 | 0 | 0 | 0 |
| *Oestrus ovis* | 0 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 4 | 3 | 0 |
| Slaughtered | 4–30 | 9–31 | 3–30 | 9–31 | 9–31 | 7–31 | 9–31 | 8–24 | 3–30 | 3–30 | 3–30 |
| Strongyloid egg-count postmortem | 1 | 0 | 0 | 5 | 0 | 16 | 23 | 17 | 0 | 0 | 0 |

At this time they were divided into four groups, three for treatment and one for controls. Feces were taken, weights were recorded, and treatment administered every three weeks.

*Group 1:* Each animal received 5 cc of tetrachlorethylene in 2 ounces of mineral oil.

*Group 2:* Each animal received 2 ounces of a 1 per cent copper sulfate solution, to which had been added 6 minims of Blackleaf 40. The dose of Blackleaf 40 was increased each time the lambs were treated until they were getting 16 minims at each dose. When the lambs were 2 years old, the dose of Blackleaf 40 was increased to 24 minims and the copper sulfate to 3 ounces of a 1½ per cent solution.

FIG. 2. No. 3 barn, showing improved hay-racks, with partitions, from rear. Note board floor.

*Group 3:* Each animal received from 2 to 3 ounces of a 1 per cent solution of copper sulfate. When they were two years old, this was increased to 3 ounces.

*Group 4:* Used as controls. All of these animals were housed in the sheep-shed during the winter months where the specially constructed hay-rack had been installed to prevent contamination of the hay. In the summer months pasture-lot rotation was followed at 2-week intervals. The majority of these animals have been slaughtered and the postmortem results are shown in tables II and III.

*Table II:* The five animals in group 1 received tetrachlorethylene. On postmortem, one was negative of parasites; the

four others were infested to a slight degree. The six animals in group 2 received copper sulfate and Blackleaf 40. All of this group showed slight infestation.

*Table III:* The six animals in group 3 received copper sulfate solution. Four of these animals were negative of internal parasites, but two of the four showed slight infestation with *Oestrus ovis*. The remaining two in this group were infested to a slight degree, with internal parasites.

The eight animals in group 4 (controls) did not receive any medicinal treatment. Three of these animals did not show any parasites on postmortem. The five others were very slightly infested.

A summary of the postmortem results of these four groups would seem to indicate that the parasitic infestation had been

TABLE III—*Postmortem findings.*

| | GROUP 3 | | | | | | GROUP 4 | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Treatment | 3 oz. 1½% copper sulfate solution every 3 weeks | | | | | | Controls | | | | | | | |
| Sheep | 7 | 8 | 15 | 16 | 44 | 14 | 17 | 19 | 23 | 24 | 25 | 26 | 28 | 34 |
| Parasite | Number Found Postmortem | | | | | | | | | | | | | |
| *Ostertagia circumcincta* | 0 | 0 | 0 | 0 | 0 | 28 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Haemonchus contortus* | 0 | 0 | 0 | 10 | 0 | 1 | 10 | 0 | 1 | 0 | 0 | 0 | 1 | 12 |
| *Nematodirus filicollis* | 0 | 0 | 0 | 0 | 0 | 17 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Oesophagostomum columbianum* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Bunostromum trigonocephalum* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Trichuris ovis* | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| *Cooperia curticei* | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Trichostrongylus extenuatus* | 0 | 0 | 0 | 0 | 0 | 21 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *Oestrus ovis* | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 |
| Slaughtered | 3–30 | 1–29 | 3–30 | 4–30 | 8–31 | 9–31 | 7–31 | 7–31 | 3–30 | 3–30 | 2–30 | 7–31 | 2–30 | 3–30 |
| Strongyloid egg-count postmortem | 0 | 12 | 0 | 0 | 2 | 3 | 18 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |

controlled to a great extent. In group 4 (controls) the degree of infestation was no greater than in any of the other groups.

Therefore it would seem that the special hay-rack and other sanitary measures were effective in preventing contamination of the feed with larvae, thus controlling infestation in the several groups comprising the flock.

The lambs of 1929 and 1930 have been raised by the same system, and examinations of the feces indicate a very slight infestation, comparing favorably with the records of the 1928 lambs.

The 1931 lambs are being raised in practically the same manner, but more attention has been given to care and sanitation. The majority of these lambs were born in March and April, and they were turned to pasture May 19. They were all negative to feces examination until July 7, at which time two animals showed a few eggs in their feces. On July 22 another examination of the feces was made and no eggs were found.

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## U. S. Civil Service Examination

The United States Civil Service Commission annonuces an open competitive examination for junior veterinarian to fill vacancies in the Bureau of Animal Industry, Department of Agriculture, Washington, D. C., for duty in the field, and in positions requiring similar qualifications. The department wishes men for these positions. The entrance salaries range from $2,000 to $2,600 a year.

Competitors will be rated on theory and practice of veterinary medicine, and on veterinary anatomy, physiology and pathology, and meat inspection. Graduation from a veterinary college of recognized standing is a requirement.

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than May 12, 1932.

Full information may be obtained from the Secretary of the United States Civil Service Board of Examiners at the post office or custom-house in any city, or from the United States Civil Service Commission, Washington, D. C.

# SOME BIPOLAR ORGANISMS FOUND IN PNEUMONIA IN SHEEP*

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On the assumption of a host specificity it has become customary to classify the organisms of the hemorrhagic septicemia group according to the animal in which they are encountered. Thus *Pasteurella avicida*, *P. boviseptica*, *P. suiseptica*, etc., are standard names in American literature. Many workers have attempted to find biochemical and serological bases which would be valid within the species or even transcend them. Much of this work has been contradictory and confusing.

Most of the recent work goes back to Jones[1] who, in 1921, found both a biochemical and a serological relationship by which he set up three groups in the bovine species. Briefly his group I organisms fermented lactose, maltose and mannitol, did not produce indol, were hemolytic and non-virulent. Group II failed to ferment lactose, maltose and mannitol, produced indol, were non-hemolytic, bile-soluble and relatively non-virulent. Group III were like group II except that they fermented mannitol, were not bile-soluble and were virulent for rabbits.

In the main, these groupings were retained when agglutinating sera were applied.

Roderick,[2] by means of complement fixation, was able to differentiate a bovine-swine group from an ovine-avian-rabbit-cavia type.

Fitch and Nelson,[3] were unable to show any striking fermentative differences in the organisms with which they worked but separated out four well-defined groups on the basis of agglutination.

Cornelius[4] collected 26 strains from different animals and found that agglutinatively they fell into four groups without regard to their animal origin.

Edington[5] found that all of the organisms which he isolated from certain cases of bovine pneumonia, both biochemically and serologically, belonged to Jones' group I. He collected a large number of pasteurellae from other laboratories, coming from a number of different animals. These fell into Jones' group III

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when tested biochemically but showed little antigenic relationship when tested with agglutinins. He found no group II strains. Eddington added mannose to the differential sugars and found that group III strains did not grow on McConkey's bile agar.

The only worker who seems to have paid specific attention to strains from sheep was Spray,[6] who isolated a large number of cultures from cases of pneumonia in those animals. He found three groups with a number of variants in each. His *P. ovisepticum* (s8 type) comprised the classical pasteurellae and would fall into Jones' group III if Jones' grouping may be applied to sheep strains. His second and third groups he classes as maltose-fermenting strains, differing from the true pasteurellae not only in the fermentation of maltose but in their inability to produce indol and in being hemolytic on blood-agar plates. They differed from each other in their fermentation of lactose and in the type of growth in glycerol-serum broth. While technical difficulties were encountered in the application of the agglutination tests, Spray felt that he had confirmed the above grouping serologically

## Sources of Cultures

The strains on which this study is based were isolated in this laboratory on rabbit-blood agar, either directly or by rabbit inoculation, from cases of pneumonia in sheep and cattle.

As will be seen by reference to the detailed history of the cultures, very few came from the same premises and in many instances the tissues from which they were derived came from widely separated points in the state. For comparison one strain isolated by us from a typical outbreak of fowl cholera and one from a case of rabbit pneumonia are included. The cultures labelled 1336 and 4277 were kindly furnished by Dr. F. S. Jones, of the Rockefeller Institute, and came from cattle. His 1336 is a typical pasteurella and belongs to his group III, while 4277 is representative of group I.

### Sheep Strains

| | |
|---|---|
| 7 | Feeder lamb, isolated from rabbit, inoculated subcutaneously with lung emulsion, 10-29-29. |
| 9A | Feeder lamb, isolated from lung direct, 10-24-29. |
| 11A<br>11B | Feeder lamb. A, isolated from lung direct; B, from inoculated rabbit, 10-29-29. |
| 18A<br>18B | Feeder lamb. A, isolated from rabbit, inoculated subcutaneously; B, from rabbit inoculated intraperitoneally, 11-4-29. |
| 19B | Feeder lamb, isolated from inoculated rabbit, 11-4-29. |
| 20A | Feeder lamb, isolated from lung direct, 11-8-29. |
| 24 | Sucking lamb, 3 weeks old, direct culture, 3-29-30, Windsor, Colo. |
| 25 | Sucking lamb, 1 month old, direct culture, 4-12-30, Montrose, Colo. |

| | |
|---|---|
| 26 | Sucking lamb, 1 month old, direct culture, 4-12-30, Akron, Colo. |
| 27 | Breeding lamb, 6 months old, direct culture, 9-26-30. |
| 30 | Feeder lamb, direct culture, 11-28-30. |
| 31 | Feeder lamb, isolated from inoculated rabbit, 11-29-30. |
| 32 | Feeder lamb, direct culture, 11-30-30. |
| 33 | Breeding lamb, direct culture, 12-2-30. |
| 34B | Feeder lamb, isolated from inoculated rabbit, 12-3-30. |
| 36 | Sucking lamb, 6 weeks old, direct culture, 2-27-31. |
| 37 | Sucking lamb, 4 weeks old, direct culture, Monte Vista, Colo., 5-12-31. |
| 38 | Sucking lamb, 3 weeks old, direct culture, Monte Vista, Colo., 5-15-31. |

CATTLE STRAINS

| | |
|---|---|
| Hayden | Isolated from outbreak of pneumonia in dairy cows at Arvada, Colo., in 1928. |
| Kingman | Isolated from outbreak of pneumonia in feeder calves, 1929. |
| Nelson | From pneumonia in feeder steers at Kiowa, Colo., summer 1930. |
| Savage | From outbreak of pneumonia in calves at Colorado Springs, that had just been vaccinated against blackleg. |
| Maynard | From pneumonia in feeder steers, fall 1930. |
| Scott | From calf showing pleuropneumonia, fall 1930. |

## BIOCHEMICAL STUDY

Carbohydrate fermentation was carried out in Wasserman tubes containing approximately 1 cubic centimeter of a meat extract bouillon (pH 7-6) to which had been added 1 per cent of the appropriate sugar and brom cresol purple as the indicator. Glycerol was used in 5 per cent solution. The cotton plug was filled with paraffin to prevent evaporation and incubation at 37°C, was carried out for 30 days. It was soon discovered that heat changed some of the sugars, so that arabinose, dextrin, dulcitol, inositol, maltose, mannitol, mannose, raffinose and sorbitol were sterilized by filtration.

Indol was determined in Dunham's peptone after incubation for 48 hours by the Ehrlich-Bohme technic, as described in the Manual of Methods, Society of American Bacteriologists. Hemolysis was measured by surface streaking on rabbit-blood agar plates. Rabbit virulence was shown by the addition of a few cubic centimeters of sterile .85 per cent salt solution to a culture on a blood-agar slope and injecting 1 cubic centimeter of the heavy suspension intraperitoneally.

## DISCUSSION OF THE BIOCHEMICAL RESULTS

The classification into typical and atypical strains is made on the basis of indol-hemolysis-rabbit virulence because these characters seem so clearly marked and in our hands unchangeable. Cultures were not easily carried even on rabbit-blood agar, sometimes dying suddenly without known cause but they did not lose virulence in any reasonable time. The cultures were transferred every 30 days and stored in a refrigerator. This

seemed satisfactory until recently, when we lost strains 20A and 30.

No culture produced gas from any carbohydrate. It will be seen that dextrin, inositol and maltose are as differential as the indol-hemolysis-rabbit combination, thus giving six characters which seem quite firmly established. All strains could be placed on this basis except two, 34B, a sheep strain, and Nelson, a culture from cattle. It may be expected that no classification can include all variants. No originality is claimed for these findings

TABLE I—*Biochemical characters of Pasteurella organisms from sheep.*

| | Arabinose | Dextrin | Dextrose | Dulcitol | Galactose | Glycerin | Inositol | Inulin | Lactose | Levulose | Maltose | Mannitol | Mannose | Raffinose | Rhamnose | Salicin | Sorbitol | Sucrose | Xylose | | Indol | Hemolysis | Rabbit |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **Typical Strains** | | | | | | | | | | | | | | | | | | | | | | | |
| Sheep | | | | | | | | | | | | | | | | | | | | | | | |
| 7 | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 11A | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 11B | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 18A | − | − | + | − | + | + | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 18B | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 19B | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 25 | + | − | + | + | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 31 | − | − | + | − | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| 33 | − | − | + | − | + | + | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| Cattle | | | | | | | | | | | | | | | | | | | | | | | |
| Scott | − | − | + | − | + | − | − | − | − | + | − | + | + | − | − | − | + | + | − | | + | − | + |
| Hayden | − | − | + | − | + | − | − | − | − | + | − | + | + | + | − | − | + | + | + | | + | − | + |
| Jones 1336 | − | − | + | − | + | − | − | − | − | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| Other Animals | | | | | | | | | | | | | | | | | | | | | | | |
| Rabbit | + | − | + | + | + | − | − | − | + | + | − | + | + | − | − | − | + | + | + | | + | − | + |
| Fowl | + | − | + | + | + | − | − | − | − | + | − | + | + | + | − | − | + | + | − | | + | − | + |
| **Atypical Strains** | | | | | | | | | | | | | | | | | | | | | | | |
| Sheep | | | | | | | | | | | | | | | | | | | | | | | |
| 9A | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 20A | − | + | + | − | + | − | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 24 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 26 | − | + | + | − | + | + | + | − | − | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 27 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 30 | − | + | + | − | + | − | + | − | − | + | + | + | + | − | − | − | + | + | + | | − | + | − |
| 32 | − | + | + | − | − | − | + | − | − | + | + | + | + | − | − | − | + | + | + | | − | + | − |
| 36 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 37 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| 38 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| Cattle | | | | | | | | | | | | | | | | | | | | | | | |
| Kingman | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| Maynard | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| Savage | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| Jones 4277 | − | + | + | − | + | + | + | − | + | + | + | + | − | + | − | − | + | + | + | | − | + | − |
| Unclassified | | | | | | | | | | | | | | | | | | | | | | | |
| 34B | − | + | + | − | + | + | + | − | + | + | + | + | + | + | − | − | + | + | + | | + | + | + |
| Nelson | − | − | + | − | + | + | − | − | − | + | − | + | − | + | − | − | + | + | + | | − | − | − |

since Jones, Spray and Edington have observed similar phenomena.

All of the cultures fermented mannitol and were insoluble in bile so that none could be placed in Jones' group II. Even though Jones' group II is more prevalent than now seems to be the case, it could be considered as merely a variant of his group III or of the typical pasteurella.

Other workers have stressed lactose as of differential importance but the rabbit culture, which is a typical pasteurella, readily fermented this substance, whereas atypical strains 26, 30 and 32 never did. Glycerol also was found useful by Spray, but in our hands glycerol fermentation was slow and uncertain.

Arabinose, dulcitol. mannose and raffinose may be of value in separating out sub-groups. None of the atypical strains fermented arabinose, while of the typical strains the three bovine cultures did not, but six of the sheep organisms and those from the fowl and rabbit did. It is interesting that dulcitol fermentation parallels arabinose throughout.

Mannose would have differentiated the two groups as found by Edington but for sheep cultures 30 and 32, which agglutinatively also form a sub-group. Raffinose is fermented by the atypical strains with the exceptions of 30 and 32. Only two of the typical cultures, Hayden and Fowl, react with this substance.

Previous workers have suggested that a group so different from the classical organisms as the atypical strains seem to be should be designated by a separate species name. In conformity with this view we suggest the name *Pasteurella hemolytica* for the group that are hemolytic, do not produce indol, are non-virulent for rabbits and produce acid in unheated dextrin, inositol and maltose.

## Serological Study

Rabbits were injected with representative cultures until a reasonably high agglutinating titre was obtained. Cultures were grown on rabbit-blood agar plates for 48 hours and then washed off in physiological salt solution. Usually the rabbit received 0.5 cc intraperitoneally at the first injection. The second was 1 cc in the same manner, the third 1 cc intravenously, and the fourth 2 cc intravenously. After this, all injections were intravenous and were of 2 cc of the heavy suspension. The interval between injections was 3 days. A satisfactory serum could be produced in from 60 to 75 days although a higher titre could be obtained with the typical cultures. The atypical strains were

used without heating or the addition of phenol, while with the typical strains 0.5 per cent phenol was added and in the later work this was followed by heating in the water-bath at 60°C. for 30 minutes. It was found that phenolization was effective in destroying the virulence of the organisms only after several days. As a result of the destruction of several partially immunized rabbits by newly prepared antigen, phenolized for too short a time, heating of all of the virulent organisms prior to injection was adopted. Several rabbits were killed with freshly phenolized antigen after having received as many as ten injections of the killed cultures. This is significant from the standpoint of the value of killed organisms when used as vaccines.

Several check tests were made that do not appear in the record, as only those that seem necessary to indicate groupings are presented. All, however, pointed in the same direction and tended only to confirm the groupings here shown. All serums were run against all antigens but to conserve space only those showing positive results were given.

TABLE II—*Serological grouping of typical strains.*

| ANTIGEN | DILUTIONS | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 50 | 100 | 200 | 500 | 1000 | 2000 | 10,000 | 20,000 |
| **SERUM FROM SHEEP CULTURE 7** | | | | | | | | |
| 7 | + | + | + | + | + | + | + | + |
| 11A | + | + | + | + | + | + | − | − |
| 11B | + | + | + | + | + | + | − | − |
| 18B | + | + | + | + | + | + | − | − |
| 19B | + | + | + | + | + | + | − | − |
| 25 | + | + | + | + | + | ± | − | − |
| Scott | + | + | + | ± | − | − | − | − |
| Fowl | + | + | + | + | − | − | − | − |
| Rabbit | + | + | + | + | + | + | − | − |
| **SERUM FROM SHEEP CULTURE 31** | | | | | | | | |
| 31 | + | + | + | − | − | − | − | − |
| 33 | + | + | ± | − | − | − | − | − |
| Jones 1336 | + | + | − | − | − | − | − | − |
| **SERUM FROM JONES CATTLE CULTURE 1336** | | | | | | | | |
| Jones 1336 | + | + | + | + | + | + | + | + |
| Hayden | + | + | + | + | + | + | + | − |
| 31 | + | + | − | − | − | − | − | − |
| 33 | + | + | − | − | − | − | − | − |

Note: 18A was agglutinated by no serum.

TABLE III—*Serological grouping of atypical strains.*

| ANTIGEN | DILUTIONS | | | | | |
|---|---|---|---|---|---|---|
| | 50 | 100 | 200 | 500 | 1000 | 2000 |
| **SERUM FROM SHEEP CULTURE 24** | | | | | | |
| 24 | + | + | + | + | + | + |
| 9A | + | + | + | − | − | − |
| 20A | + | + | + | − | − | − |
| 27 | + | + | + | − | − | − |
| 34B | + | + | + | + | − | − |
| 36 | + | + | + | − | − | − |
| 37 | + | + | + | + | ± | − |
| 38 | + | + | + | + | − | − |
| Kingman | + | + | + | − | − | − |
| Maynard | + | + | + | + | − | − |
| Jones 4277 | + | + | + | − | − | − |
| **SERUM FROM KINGMAN CATTLE CULTURE** | | | | | | |
| Kingman | + | + | + | + | ± | − |
| 9A | + | + | + | − | − | − |
| 20A | + | + | ± | − | − | − |
| 24 | + | + | + | − | − | − |
| 27 | + | + | + | ± | − | − |
| 34B | + | + | + | + | − | − |
| 36 | + | + | + | − | − | − |
| 37 | + | + | + | + | − | − |
| 38 | + | + | + | + | − | − |
| Maynard | + | + | + | + | − | − |
| Jones 4277 | + | + | + | + | − | − |
| **SERUM FROM SHEEP CULTURE 30** | | | | | | |
| 30 | + | + | + | + | + | + |
| 32 | + | + | − | − | − | − |

Note: 26, Savage and Nelson were agglutinated by no serum.

## DISCUSSION OF THE SEROLOGICAL RESULTS

From the tables it will be seen that with the exception of 18A the typical cultures fall into two sub-groups. The first group includes cultures from four different animals: sheep, cattle, fowl and rabbit.

The second group includes two sheep cultures (31 and 33) and one cattle culture (Hayden). Here also belongs Jones' cattle culture (1336). While 31 serum did not react with Hayden antigen it seems probable that it would have done so had a higher titre been produced. Most of the atypical strains fall into a single group. Only sheep and cattle strains are found here, as in this series there were no atypical strains from other animals. From the work of others, however, we may believe that they exist. The sheep culture, 34B, that could not be classified bio-

chemically, falls in with the atypical group when tested with agglutinating serums.

Cultures 30 and 32 seem to form a sub-group both biochemically and serologically. Their fermentation of mannose and their inactivity on raffinose separate them from the other atypical strains. The fact that 30 serum, with a titre of 1-2000, agglutinates 32 antigen to only 1-100 indicates that they are somewhat removed from each other. Sheep strain 26 and cattle strain Savage were not grouped serologically. Biochemically they belong with the 24-Kingman division, but they were agglutinated with no serum with which they were tried. Nelson, a cattle culture, could not be grouped either biochemically or serologically.

## SUMMARY

A biochemical and a serological study was made of 20 strains of bipolar organisms isolated from cases of pneumonia in sheep and 6 from pneumonia in cattle.

For comparison representatives of Jones' groups I and III were used as well as a culture from a fowl and one from a rabbit.

On the basis of indol, hemolysis and rabbit virulence, all cultures could be separated into two groups except two, one a cattle culture and one from a sheep.

If this method should be found applicable to strains from all animals, then Jones' group I of the bovine type falls in with the atypical organisms and his group III with the typical or classical pasteurellae.

It is recommended that the atypical group be given a species name, *Pasteurella hemolytica*.

Serologically there was no cross-agglutination between the typical and the atypical organisms. These two, however, could be still further subdivided into at least two sub-groups. The serological method left four strains unplaced.

## CONCLUSIONS

Classification of the genus Pasteurella on the basis of host specificity leaves much to be desired when the organisms are tested biochemically and serologically.

The organisms isolated from sheep and cattle can be divided biochemically and serologically into at least four sub-groups that cross species lines.

It seems time to give the atypical pasteurellae (Jones' group I, maltose-fermenting strains of Spray) a species designation.

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DISCUSSION

DR. B. T. SIMMS: I would like to ask Dr. Cross if he checked various strains on different types of animal, and, if so, if he could give us a very brief discussion on this.

DR. CROSS: Atypical strains are non-pathogenic for rabbits. Jones has stated that the atypical strain is unquestionably associated with pneumonia, but it has never been isolated from the blood. Therefore, he does not feel it is a septicemic disease, as is true with the typical strains.

DR. J. F. BULLARD: What special method is used in isolating the organisms?

DR. CROSS: We isolate these oragnisms by culturing them on blood-agar plates.

## Be Kind to Animals

A special reminder to humans to be humane was the slogan cancellation, BE KIND TO ANIMALS, used on Canadian mail during National Be Kind to Animals Week, April 18-23. Apropos of this reminder is the American Humane Society suggestion that we be sure to be kind to animals always—not just for one week each year.

The United States Post Office Department, formerly a frequent user of the slogan cancellation form of national reminder, has abandoned the practice for general use because of inability to comply with the flood of requests for special slogan cancellations for various events. Only Post Office Department matters are now the subjects of these special cancellations in the United States.

## German Veterinarian Lectures at Pennsylvania

Dr. Oskar Seifried, who has been engaged in research work at the Rockefeller Institute, Princeton, New Jersey, for the past three years, lectured before the students of the School of Veterinary Medicine, University of Pennsylvania, on March 3, 1932. His subject was "Histology and Cytology of Infectious Diseases Caused by Filtrable Virus." On April 1, Dr. Seifried succeeded Professor Kitt as Professor of General Pathology and Pathological Anatomy, and Director of the Pathological Institute of the veterinary faculty of the University of Munich. This appointment was announced in the November, 1931, issue of the JOURNAL.

# HOG-LOT SANITATION AND ITS RELATION TO SWINE DISEASE CONTROL*

*By* H. A. WILSON, *Jefferson City, Mo.*
*State Veterinarian*

If historians are correct, when grandfather was a boy, hog diseases were not a source of worry to the average hog-raiser. It is claimed that hog cholera did not make its appearance in the United States until the year 1833, slightly less than one hundred years ago.

If hog cholera is an ancient disease, it was either little recorded or else did not assume such a vicious role as it does today. The mere fact that we have no history of cholera outbreaks on the American continent prior to 1833 would lead us to believe that hog cholera is not an old disease, as we do know that we had hogs on the North American Continent for approximately three hundred years before the first outbreak of cholera in the Miami Valley of Ohio, which was presumably imported from Europe.

De Soto is credited with being the first importer of swine to what is now the United States of North America—a historical fact, I believe, which has never been successfully disputed. De Soto did not have to worry about raising his hogs on clean ground (they had an unlimited range), and did not have to worry about parasites, except those which wore feathers and carried tomahawks. From this band of hogs, imported by De Soto, descended what has commonly and erroneously been called the "American wild hog," which early settlers hunted and feasted upon at every opportunity.

With the approach of civilization, and the settlement of the country by the pioneers, open range was the rule and not the exception. People did not raise hogs as they are raised today—the hogs simply raised themselves—and when butchering time arrived, the settlers sallied forth with their trusty flintlocks and brought home the year's supply of hams and bacon. As the country became more densely populated, and farms fenced, hogs were then confined in circumscribed quarters. Even under these artificial conditions, the hog, until the first outbreak of cholera, was considered a very hardy animal—so hardy that it

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

scarcely needed any attention at all on the part of the keeper, most people even entertaining the idea that a hog was immune to poison—a tradition still prevailing in the minds of some people.

Even when hog cholera made its appearance, it apparently did not cause much concern, except to kill off the hog crop periodically in certain communities.

My father was raised in the Miami Valley of Ohio—in fact, moved there with his parents some three or four years after the first outbreak of cholera occurred. The people in that section did not look upon cholera as an infectious disease, but rather considered it the wrath of the Lord, sent upon them as a punishment for their sins; and often I have heard my father say that cholera appeared only about once each decade. I merely make mention of these things in order to show you that our early farmers, even slightly less than a hundred years ago, were not confronted with such problems in connection with the raising of hogs as are modern-day American farmers.

## Hog Cholera Virus Indicted

Fortunately, about twenty-five years ago, there was discovered a preventive for hog cholera, but, unfortunately, at the same time, there was turned loose in the United States an agent which has been indisputably the greatest spreader of hog cholera known to the civilized world. That agent is hog cholera virus, which is indiscriminately distributed throughout the land in practically every state in this Union. I am firm in my convictions that hog cholera virus has not only done a thorough job of distributing hog cholera throughout the United States, but, at the same time, it has also been a potent vehicle for the distribution of other infections which are now causing veterinarians and hog-raisers great concern.

I have just finished saying that we have a preventive for cholera, and, therefore, my remarks may seem inconsistent. Yes, we have the preventive, but it is not always intelligently used, and certainly it is not restricted entirely to trained hands. Furthermore, many a good drove of hogs has been sacrificed with the cholera while the expert was chasing some other bug (who happened to be an innocent bystander) down the ravines and over the hills, when said bug, if let alone and the herd properly treated, need have caused no more concern than a louse on a dead "lumberjack."

It is not my desire to leave the impression that cholera is the only disease of hogs, for I am not overlooking hemorrhagic septicemia, as well as a few others which have been definitely and intelligently recognized. However, I do think that we are calling a lot of complications of such diseases as cholera, hemorrhagic septicemia, and even conditions like parasitism "diseases," and attempting to segregate them and treat them individually, and while doing so, actually overlooking the fundamental disease or condition which is causing the farmer monetary loss not only in mortality but in unthriftiness.

By the process of common reasoning, we ought to know that the hog, or any other animal, when kept under what might be considered artificial conditions, is subjected more and more to the abundant supply of bacteria and other infective organisms year in and year out, and that in the due course of time the body of the hog, or other animal, will do one of two things, namely: lose its resistance to the invasion of these organisms, or else build within its body a greater resistance which will enable it at least to tolerate them. I might venture the theory that the American hog family at the present time has just reached the stage where its resistance is broken, as that may explain why we are confronted with so many so-called diseases at this time.

Scientists tell us there are two main ways of coping with disease, as far as prophylaxis is concerned: One is total avoidance; the other is through the medium of some immunizing agent.

If we adopt the modern theory that we have a large number of hog diseases, then, unfortunately, we have only one trustworthy immunizing agent—anti-hog cholera serum. Consequently, we are groping in the dark in handling most of the other diseases, and it appears to me that the sensible course to pursue is to adopt the principle of avoidance instead of adhering to the old rule of locking the stable door after the horse is stolen.

### McLean County System of Swine Sanitation

I am at a great loss to understand why the veterinary profession has not taken more kindly to the so-called McLean County System of Swine Sanitation, and have not set their heads to learn as many details of the system as is humanly possible, and then devoutly advocate its adoption on the part of their clientele. To my utter amazement, I occasionally find a veterinarian who even condemns the system, and goes as far as to tell his clients that he does not believe there is much to it. Such action

on the part of an individual veterinarian is excusable on only two grounds: One is ignorance; the other is blind prejudice toward the system because it is being advocated by extension workers throughout the land.

In experiments conducted by the Extension Department of the University of Missouri, it was demonstrated on farms that where a hog-owner practiced all of the things he was told to do he obtained eighty per cent perfect results; whereas, the fellow who practiced only one of the many things he was told to do obtained only three per cent perfect results. Suffice it to say that there must be a missing link in the chain of the system or else near one hundred per cent perfect results could be obtained.

I think the weakest link in the chain is the contamination of the sow herself with intestinal parasites when she is placed in the farrowing-pen. In other words, if the sow is positively worm-free, and then given a good external cleaning and placed in a properly cleaned and disinfected pen, the young offspring will have no chance whatever of obtaining any worm eggs during their first days of life. Doubtless another element entering into the twenty per cent failure is inefficiency, or lack of knowledge, on the part of the hog-raiser when preparing the sow for the farrowing-pen—another argument in favor of keener interest and closer study on the part of the practitioner in connection with the system, in order that he can pass it on to his clients, much to their advantage.

### Effective for Other Internal Parasites

We, of course, must admit that the so-called McLean County System has been directed mainly toward control of the ordinary roundworm, or, to be more scientific, the *Ascaris lumbricoides;* yet, recent experiments by government parasitologists, I believe, have demonstrated that the system is equally effective in controlling other forms of internal parasites, and doubtless will work equally as effectively in controlling certain filth-borne diseases.

I think the rotation of hog-lots of itself is a splendid idea, and cannot help but remove the hog from the environment of many forms of bacteria and other low forms of life, which of itself is conducive to more profitable hog-raising. It is hard for one to imagine a more insanitary place than an old hog-lot, which has been used for generations for the raising and feeding of hogs. Theoretically, it should be a botanical garden for bacteria and a zoo for parasites. Cultivation of soil has some unknown puri-

fying property; while subjecting soil to certain crops only occasionally seems to control certain plant diseases. The best illustration of this is tomato blight, as tomato-raisers recognize that to continue growing the crop on the same ground puts them out of business. Why should not the same rule apply in the raising of hogs as well as other animals?

We must not, and should not, expect too much of the McLean County System, or any other system, for there will always be loopholes and leaks to be stopped; something will be left undone that will tend to disrupt and interfere with the plan. We North Americans have too much of a tendency to discover a thing, then turn it over to the mercy of the public, and spend our time hunting new fields for adventure. I would like to see more research work done along the line of disease prevention by the modern sanitary plan of raising hogs, and see if some other of the present mysteries can be unraveled, as to do so will undoubtedly place hog-raising upon a more satisfactory basis. It will sift the wheat from the chaff when it comes to hog diseases, and perhaps eliminate many things which are now looked upon as diseases, which in reality are nothing more nor less than harmless interlopers.

### Practitioners Should Keep Posted

Practitioners should avail themselves of all available literature upon the subject of swine sanitation; they should give the matter deep study, and advocate only such practices which in their own minds they know that the people of their community can and will adopt. There is no use in advocating or advising the farmer or any other man, to do a thing which he cannot do. Plans must be altered to suit the occasion. It is folly to advise some poor tenant farmer to install concrete feeding-floors on another man's land, which is leased only from year to year. Likewise, it is greater folly to preach hog-lot rotation to a man farming under similar curcumstances who has only one hog-tight fence on the farm. Nevertheless, those men have their problems to solve, and if the practitioner puts his heart and soul into the business, he can make some suggestion which that type of a farmer can adopt, and which will enable the farmer to raise hogs at a profit instead of at a loss.

In conclusion, in making apologies for taking up your time with this paper, I want to leave two thoughts in your mind: One is that the veterinary profession today is too busily occupied in

looking at lesions, which of course can be directly traced to certain organisms, but which in many cases are secondary, thus overlooking the fundamental cause of the sickness—and I do not hesitate to say that, alas, in too many cases it is nothing more nor less than some form of hog cholera, even though it occurs in a so-called immunized herd, while in other cases the morbid condition is nothing more nor less than the effects of super-saturation of filth, with its attending evils, on the part of the herd in question.

### Change of Food Brings Improvement

We frequently see the picture of a bunch of hogs gone wrong. This practitioner is called, and that one is called. Then some agency of either the state or federal government is appealed to for the services of an individual commonly designated as an "expert." When the expert arrives on the scene, if perchance he does not stump his toe and fall down over something that the local citizens have failed to recognize, he is just as helpless as a cowboy navigating on the high seas. About the only thing that he can suggest is, of course, to send some of the specimens to a laboratory and then a change of pasture. The specimens go to the laboratory where they are carefully examined and cultured. Few laboratories have the facilities to care for the large number of test-tubes of cultures after they are isolated, for many hideous-looking bugs are found. In the meantime, if the owner has followed the instructions of the expert, and the hogs are placed in a clean pasture, unless perchance it is a plain case of cholera, a rapid improvement is soon noted, and of course the expert gets the credit.

I am not belittling the experts, for we certainly have experts in many lines of work, although I can truthfully say that, with the possible exception of liars, I have never seen one that ran true to form, and deserved the honors. Now the moral to this hypothetical case is that if those hogs had been intelligently handled, and switched about on clean ground from time to time, the owner would not have had these troubles. Again, if the owner had not switched them around, and his troubles were brought on by his own acts of carelessness and indifference, then the first practitioner called should have advised a change of pasture.

The other thought is that any system, which, when diligently followed by an untrained man, will bring eighty per cent perfect

results as compared with the old plan (or, rather, lack of plan), certainly has virtue, and is undoubtedly a most potent and effective weapon to be used in the avoidance of a lot of hog grief listed under different headings; and when the veterinary profession, including government men, state men, and practitioners, makes a closer study and takes a more profound interest in such a system, many of our present-day troubles will vanish. Furthermore, practitioners, individually and collectively, should be advocating the system more generally, and should not leave it to some paid employe, either from the extension service or some other service, perhaps in the employ of some commercial hog-remedy company, to come into his community and show the very people he is dependent upon for a livelihood a better way to raise hogs, as, to express it mildly, that is a very poor means for the practitioner to intrench himself in the hearts and minds of his people.

Again I wish to state that in my mind the average practitioner is more capable and better qualified to present these things to his people than is any outside individual, as, if he does not, he ought to know the problems of his community as no other man could know them.

### Discussion

Dr. J .L. Jones: Dr. Lytle sort of put me in a "crack" when he asked me to discuss Dr. Wilson's paper, because it happens we were classmates, live in the same state, came from the same county, and practiced for a number of years in adjoining towns, and so naturally might see things in the same light. In spite of that, we don't always see them in the same way, and probably most of the time Dr. Wilson is right when we don't.

I want to endorse heartily the McLean County System. I believe veterinarians engaged in swine disease work, whether from the standpoint of a state official or from the standpoint of a practicing veterinarian, should at all times be ready and take every opportunity to advise the swine-owner regarding his sanitary problems. In my practice, I consider that is a part of my job. I never lose an opportunity to discuss that with my clients. The trouble is that you can't always get one hundred per cent coöperation; in fact, you can't get as good as Dr. Wilson mentioned—eighty per cent results.

Since that is true, and since it is a fact that we do have a large number of insanitary hog-lots where animals are kept, we probably do have a great many diseases to contend with that ordinarily would be eliminated. I do believe that in spite of the McLean County System, or any other system of sanitation, we will have outbreaks of contagious diseases among swine rather frequently in sections where a number of swine are grown or where there are a number of large herds.

I have in mind, at the present time, a herd of hogs I was called to see last week, owned by a man considered to be one of the foremost hog-raisers in my community, who is a believer in the McLean County System, and tries to carry it out to the best of his ability, and really does a pretty good job of it, I think. At least, he has made a sincere effort to do so.

It so happened that one of his neighbors had been having some sick hogs. This man had a herd of pigs weighing around thirty pounds that had not been vaccinated. This man, in an effort to save some money, as every one is trying

to do at the present time, had some serum sent down from a certain laboratory and vaccinated his hogs. At the last account, thirty-five of them had died.

This client of mine, with the nice pigs, trying to follow the McLean County System, had a good percentage of his herd sick when I was called out there. So those things do happen.

My friend, Dr. Wilson, is of the opinion that if we could control hog cholera and watch sanitation, most of the other conditions would control themselves. I will have to take issue a little with that statement. We have discussed it before on the floor. We have found hemorrhagic septicemia in hogs that have been vaccinated and in those that have not been. I know of instances where that diagnosis has been made and we have gone in and used the biological products we ordinarily used—bacterins—and controlled that condition without using any hog cholera serum at all. I am quite sure the diagnosis was correct and that the treatment was correct.

I do believe it is up to us to try to educate the hog-owner with respect to sanitation in so far as possible, but we must not lose sight of the fact that hogs have other diseases than hog cholera. In Missouri, at the present time, we will find hogs infected with necrotic enteritis. That is a filth disease. We have hemorrhagic septicemia.

There are many people like a farmer I heard about who, upon seeing a giraffe in a circus, said, "There ain't no such animal." Many people say that about pulmonary edema. Whether pulmonary edema is a specific disease is questioned. In Missouri we have had hogs infected with a condition we termed that and we have used autogenous bacterins and they have given results.

Dr. Wilson mentioned the discovery of anti-hog cholera serum some twenty-five years ago. Personally, I think that is the greatest boon the hog industry ever had, but at the present time hog cholera virus is being distributed indiscriminately, and I do believe there is a great deal of work to be done on the control of the distribution of virus. It is being put into untrained hands and in many instances is doing much damage. I beleve you gentlemen can do a great deal of work in that direction, that is, the control of the distribution of virus.

CHAIRMAN CREWE: In Dr. Jones' closing remarks, he brought out an extremely vital point and that is in regard to the control of the distribution of virus. In North Dakota, for some years, hog cholera was comparatively unknown. We realized that, when the industry grew, eventually these diseases were going to come in. We tried to restrict them in every way possible. We restrict the use of hog cholera virus to veterinarians officially authorized to use it. In spite of everything we can do, there are certain serum-producers who will bootleg virus to the laymen. They ship it in and we find it almost impossible to get any service on them or prosecute them or prevent the practice, and we doubt if it will ever be done until the federal government establishes some means of controlling it. I do believe that it is a very vital problem.

DR. WILSON: I don't want to leave the impression that I think hog cholera is the only disease of hogs. It would be silly for any one to take that attitude. The human race has more than one disease: dogs and cats have more than one, and why shouldn't hogs? However, I don't believe there are any 1189 different diseases, as we sometimes find when we try to make a diagnosis. I have gone out in some of these cases as a "trouble-shooter." They didn't call me for what I knew, but called me for a "shock-absorber," and I knew it at the time. I found hogs with no less than fifteen or twenty different shots of different things.

In making different diagnoses, I follow Dr. James Law. I don't know whether you know anything about him or not, but I think he has written the only veterinary work ever written. Dig out a magazine and take out some of this ultra-modern stuff, and when you have failed to grasp it, go to Chapter IV and he will tell you all about it. He told these fellows how to make hemorrhagic septicemia aggressin.

I say only this, gentlemen—I think many of these bugs causing this and that and the other things are nothing in the world but cowbirds. You know the cowbird never builds a nest: it lays its eggs in other birds' nests. They are dropping around here and there. They congregate in droves for only a short time of the year and after that they are scattered out with blackbirds and

others. I think many of these diseases are like a lot of other things—just like the fellow who goes out rabbit-hunting and keeps looking in the tops of the trees where squirrels are.

I think hog cholera is our most serious menace to the hog-lot. We will have hemorrhagic septicemia, necrotic enteritis, and pulmonary edema—if there is such a thing—but I am firmly convinced that it is invariably a side-chain with something else.

I think the first thing a veterinarian ought to do when he goes into a drove of sick hogs is to eliminate absolutely, if possible, any possibility of cholera before he goes to tinkering with anything else. I think that is only a common sense thing to do. You have all seen hogs die where the mortality ran eighty-five and ninety per cent. If you pick up any writer on the subject, you will find he makes a positive statement that the mortality is around eighty to ninety per cent, and hemorrhagic septicemia runs around ten, twelve and fifteen per cent, if left alone and nothing done with it. So what can we assume, when we see a man lose the majority of his herd, but that it is cholera? We can not reasonably and intelligently figure it out any other way.

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## A Great Benefactor of the Human Race

Fifty years ago this month Dr. Robert Koch of Germany made a discovery which has proved to be one of the greatest contributions to human welfare and happiness. He succeeded in isolating the germ causing tuberculosis. He demonstrated that a tiny rod-shaped organism multiplying in human bodies produced ailments which accounted for about one-seventh of the mortality of his time.

Koch discovered no cure for the disease and half a century of tremendous achievements in medical science failed to produce such a specific. And yet, acting upon the knowledge which Koch's discovery furnished, science and diligence of health authorities have greatly reduced the tuberculosis death rate.

The great value of Koch's discovery proved to be in the field of accurate diagnosis with the consequent possibility of combating the disease in its earliest manifestations. And out of this increase of knowledge, also, has come recognition of the importance in dealing with the disease, of providing correct housing, proper environment, rest and proper food.

Not the least of these accessory discoveries has been that bearing upon safeguarding the milk supply. So important has this proved that the one factor of tuberculin testing of cattle is counted one of the chief influences contributing to the lessened virulence of tuberculosis over large areas.

The name of that modest country doctor, Robert Koch, surely deserves enrollment among those of great benefactors of the human race.

Editorial in Detroit, Mich., *News*.

# STUDIES ON CANINE DISTEMPER:

## I. The Bacteriology of One Hundred Naturally Infected Cases*

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### INTRODUCTION

Prior to 1905, when Carré[1] reported the results of experiments in which he was able to transmit the disease by means of filtrates of serous discharges, numerous organisms were suggested as causative factors of canine distemper. From this work, it appeared for a time that the etiology of the disease was settled and a filtrable virus was then believed to be the causative factor.

A divergence of views as to the cause of distemper was brought about by the reports of Ferry,[2,3] in Detroit; McGowan,[4] in Scotland, and Torrey and Rahe,[5] in New York, in which they considered the etiological agent to be a cultivable microörganism which Ferry named *Bacillus bronchisepticus.* This divergence of views was lessened somewhat by the work of Ferry,[6] who showed that it was possible to filter *B. bronchisepticus* through filter candles which most workers would regard as being bacteria-proof, but others still held to the view that a filtrable virus was the cause of this disease. These views were well expressed by Hardenbergh,[7] who said:

> Undoubtedly, *Bacillus bronchisepticus* has certain pathogenic properties but exhibits negative properties as compared with the positive characteristics of natural distemper. These negative properties are shown (1) by its failure to produce definite distemper in most susceptible subjects, (2) by the very slight agglutinating properties imparted to the blood serum of dogs immune to distemper (it being presumed, and capable of proof, that a majority of these animals harbored the organism) and (3) by the comparatively slight protection afforded by vaccination with the organism.

In a later publication, Hardenbergh[8] states:

> . . . . in spite of its secondary role, *Bacillus bronchisepticus* still must be considered pathogenic to a degree and as having its share in the development of the organic affections that arise in the course of the disease.

And further:

> . . . . dogs which have developed some symptoms of distemper following the administration of living cultures of *Bacillus bronchisepticus,* and recovered, still remain susceptible to virulent infection by natural means.

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

In 1926, Dunkin and Laidlaw[9,10] made their first publications embodying the results of experiments, started some three years previously under the direction of the Medical Research Council in England, in which they reported a filtrable virus as the cause of canine distemper and concluded that *B. bronchisepticus* was only a secondary invader.

Following these reports, experiments were outlined to study in our laboratories a number of naturally infected cases to determine the role of *B. bronchisepticus* as well as that of a filtrable virus in the production of the symptom complex which is ordinarily diagnosed as canine distemper.

## Cases Taken for Study

One hundred cases of canine distemper in dogs were used in these studies, most of which were in the early stages at the time of receipt at the laboratories. This number included also some which showed symptoms a few days after arrival and some susceptible pups showing evidence of the disease after exposure to infection by contact with a naturally infected dog, as well as several dogs from a kennel in which natural infection had taken place. In addition, there are also included, as normal controls, a few susceptible pups destroyed before exposure to infection.

With a few exceptions, the dogs selected for these studies were in the earlier stages of the disease, since preliminary work, as well as the results of other investigators, indicated that if the animal was in the advanced stages or had died as a result of infection prior to making cultures from the internal organs, considerable difficulty was experienced in the isolation, in pure culture, of the organisms present. This was undoubtedly due either to antemortem or postmortem invasion of bacteria from the intestinal tract.

Diagnosis of distemper was made by the presence of inappetence, the typical cough, roughened hair-coat, elevation of temperature and a serous or mucous nasal discharge which in some of the more advanced cases was mucopurulent in character. In some cases conjunctivitis with serous or mucopurulent ocular discharges was seen. Diarrhea was not a constant symptom but was quite frequently seen in the early stages of the disease in puppies. With the onset of symptoms the temperature was not found to be elevated as high as 105° F., as reported by Dunkin and Laidlaw[10] in experimental distemper in dogs, but was usually 103.0 to 103.5° F. During the periods observed, this elevation

remained fairly constant and did not show the marked rise and fall as described by the above authors. As an example of the thermal curve in uncomplicated *B. bronchisepticus* infection the daily temperature readings of D-101 are shown in figure 1. In the later stages the mucopurulent nasal and ocular discharges were seen in many cases to be quite copious and in others dried to form crusts around the eyes and nose. Respiration was difficult, due to partial occlusion of the respiratory passages with these secretions and in several cases was made more so by the presence of bronchopneumonia.

On autopsy the lesions in the internal organs were seen to vary considerably, depending somewhat on the resistance of the animal, the virulence of the infection, as well as the duration of illness prior to destruction for culturing. In the very early stages, *i. e.* after one or two days of illness, practically the only lesion seen was an inflammation of varying intensity of the mucous linings of the upper respiratory passages. In addition there would be found, in the trachea and larger bronchi, a thick, glaring, grayish, tenacious mucus. Early in the disease in puppies, catarrhal enteritis was frequently seen. In later stages, other lesions such as broncho-pneumonia, fatty degeneration of the liver, congestion of the body lymph-glands and occasionally a slight enlargement of the spleen, were noted.

The dogs selected and used for these studies were destroyed by intramuscular injection of strychnin sulfate. All dogs were taken into the laboratory for autopsy and culturing which, with few exceptions, was done immediately after death. A few animals obtained from a kennel in which an outbreak of distemper had occurred were not autopsied or cultured until some twelve to twenty-four hours had elapsed.

## Method of Examination

In preparation for autopsy and culturing, each carcass was placed on an operating-table and thoroughly washed with a 5 per cent solution of cresylone. After the skin was removed from the ventral and lateral surfaces of the neck and body, the entire muscular surface thus exposed was seared by direct application of a gas flame. The body cavities were then opened with sterile instruments. Cultures were made from the lower trachea by means of a platinum loop. Those from the lungs, liver and spleen were obtained by smearing the cut surfaces of these organs on agar in three-inch petri dishes, the pieces from each organ having

TABLE I—*Bacteriological findings in distemper dogs.*

| DOG | SOURCE | SOURCE OF INFECTION | TRACHEA | LUNG | LIVER | SPLEEN | HEART BLOOD | REMARKS |
|---|---|---|---|---|---|---|---|---|
| D-1 | | | H.S. | B.B. | Sterile | Sterile | Sterile | 1 |
| D-2 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 1 |
| D-3 | Pound | Pound | B.B. | B.B. | S.A. | S.A. | B.B. | 2 |
| D-4 | | | B.B. | B.B. | S.A. | S.A. | Sterile | 3 |
| D-5 | | | S.A. | S.A. | S.A. | S.A.,C.T. | Sterile | 4 |
| D-6 | Reared | Infected quarters | S.A.,B.B. | S.A.,B.B. | Sterile | S.A. | H.S.,B.B. | 5 |
| D-7 | | | B.B. | B.B. | B.B. | S.A. | S.A.,S.Y. | 5 |
| D-8 | | | S.A.,B.B. | B.B. | S.A. | S.A. | H.S. | 3 |
| D-9 | | | Culture lost | Culture lost | Sterile | Sterile | S.A. | 3 |
| D-10 | | | Sterile | B.B.,C.T. | S.A. | S.A. | S.A. | 3 |
| D-11 | | | B.B. | S.A.,B.B. | S.A. | S.A. | S.A. | 3 |
| D-12 | | | B.B. | B.B. | S.A. | C.T. | Sterile | 6 |
| D-13 | Pound | Pound | B.B. | B.B.,S.L. | S.A.,S.Y. | Sterile | Sterile | 1 |
| D-14 | | | B.B. | B.B.,S.A. | S.A. | S.A. | S.A. | 3 |
| D-15 | | | B.B. | B.B. | S.A. | Sterile | Sterile | 2 |
| D-16 | | | B.B.,C.T. | S.L.,S.A. | S.A. | Sterile | Sterile | 6 |
| D-17 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 7 |
| D-18 | | | B.B. | B.B. | Sterile | Sterile | B.B. | 3 |
| D-19 | Country bred | Infected quarters | B.B. | S.A. | S.A.,H.S. | S.A. | Sterile | 8 |
| D-20 | | | C.T. | B.B. | Contaminated | As Liver | As Liver | 3 |
| D-21 | Pound | Pound | B.B. | B.B. | Contaminated | B.B.,S.A. | B.B. | 9 |
| D-22 | | | B.B. | B.B. | S.A. | S.A. | S.A. | 1 |
| D-23 | Country bred | Infected quarters | B.B. | S.A. | S.A. | S.A. | Sterile | 7 |
| D-24 | | | B.B.,S.A. | S.A. | S.A. | S.A. | Sterile | 7 |
| D-25 | Pound | Pound | B.B.,S.A. | B.B.,S.A. | S.L. | S.A. | S.A. | 6 |
| D-26 | | | B.B. | B.B.,S.Y. | S.A. | Sterile | Sterile | 7 |
| D-27 | | | B.B. | B.B. | Sterile | S.A. | B.B. | 10 |
| D-28 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 10 |

TABLE I—*Bacteriological findings in distemper dogs.* (Continued)

| DOG | SOURCE | SOURCE OF INFECTION | TRACHEA | LUNG | LIVER | SPLEEN | HEART BLOOD | REMARKS |
|---|---|---|---|---|---|---|---|---|

| Dog | Source | Source of Infection | Trachea | Lung | Liver | Spleen | Heart Blood | Remarks |
|---|---|---|---|---|---|---|---|---|
| D-29 | | | B.B. | B.B.,H.S. | S.A. | S.A.,S.L. | S.A. | 7 |
| D-30 | | | B.B. | B.B. | S.A. | S.A. | Sterile | 10 |
| D-31 | | | B.B. | S.A. | S.A. | S.A. | Sterile | 1 |
| D-32 | | | B.B. | B.B. | S.A. | Sterile | S.A. | 11 |
| D-33 | | | B.B. | B.B. | S.A. | H.S.,S.A. | S.A. | 5 |
| D-34 | | | B.B.,H.S. | B.B. | H.S. | Sterile | Sterile | 1 |
| D-35 | | | B.B. | S.A.,B.B. | H.S.,S.A. | S.A. | S.A. | 7 |
| D-36 | | | B.B. | B.B.,S.Y. | S.A. | Sterile | S.A. | 6 |
| D-37 | | | B.B. | B.B. | S.A. | Sterile | Sterile | 6 |
| D-38 | | | B.B. | B.B.,S.A. | Sterile | Sterile | Sterile | 6 |
| D-39 | | | C.T. | Sterile | S.A. | S.A. | S.A. | 6 |
| D-40 | Pound | Pound | B.B. | B.B. | Sterile | S.A. | Sterile | 12 |
| D-41 | | | B.B. | B.B.,S.A. | Sterile | Sterile | Sterile | 6 |
| D-42 | | | B.B. | B.B.,S.A. | Sterile | S.A. | S.A. | 1 |
| D-43 | | | B.B. | B.B.,S.L. | S.A. | Sterile | Sterile | 1 |
| D-44 | | | Sterile | B.B.,H.S. | S.A. | Sterile | S.A. | 3 |
| D-45 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 13 |
| D-46 | | | B.B. | B.B. | S.A.,H.S. | S.A. | B.B. | 14 |
| D-47 | | | B.B. | B.B.,S.A. | S.A. | S.A. | S.A. | 2 |
| D-48 | | | No growth | No growth | S.A. | No growth | No growth | 2 |
| D-49 | | | B.B. | B.B. | S.A.,H.S. | Sterile | B.B. | 6 |
| D-50 | | | B.B.,C.T. | C.T. | C.T. | C.T. | C.T. | 15 |
| D-51 | | | B.B. | B.B.,S.A. | Sterile | S.A. | Sterile | 6 |
| D-52 | Cultures from bronchus and spleen | Not known | C.T. | None made | None made | S.A.,S.B. | None made | 16 |
| D-53 | | Pound | B.B. | B.B. | B.B. | B.B.,C.T. | B.B. | 12 |
| D-54 | Pound | Infected quarters | B.B. | B.B.,S.A. | S.A. | Sterile | S.A. | 5 |
| D-55 | | | B.B. | B.B.,S.A. | S.A. | Sterile | S.A. | 2 |
| D-56 | | | B.B. | B.B. | Sterile | S.A. | B.B. | 11 |
| D-57 | Country bred | Contact D-55 | B.B. | B.B. | S.A. | S.A. | S.A. | 11 |
| D-58 | | | B.B. | B.B. | Sterile | Sterile | B.B. | 11 |
| D-59 | | | B.B. | B.B. | Sterile | Sterile | B.B. | 11 |

TABLE I—*Bacteriological findings in distemper dogs—Continued.*

| Dog | Source | Source of Infection | Trachea | Lung | Liver | Spleen | Heart Blood | Remarks |
|---|---|---|---|---|---|---|---|---|
| D-60 | Cultures from bronchus and spleen | Not known | S.A.,C.T. | None made | None made | S.A.,H.S. | None made | 16 |
| D-61 | Country bred | Intratracheal injection B.B. | B.B. | B.B. | S.A. | H.S. | Sterile | 11 |
| D-62 | Pound | Pound | B.B. | B.B. | B.B.,H.S. | B.B.,C.T. | B.B. | 2 |
| D-63 | | | B.B. | B.B.,C.T. | S.A.,C.T. | B.B.,S.A. | B.B.,S.A. | 2 |
| D-64 | Country bred | Intratracheal injection B.B. | B.B. | B.B.,S.A. | B.B. | S.A. | S.A. | 13 |
| D-65 | | | B.B. | B.B. | S.A. | S.A. | S.A. | 7 |
| D-66 | Pound | Pound | B.B.,S.A. | B.B. | Sterile | Sterile | Sterile | 12 |
| D-67 | | | B.B.,S.Y. | B.B.,H.S. | Sterile | Sterile | Sterile | 7 |
| D-68 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 7 |
| D-69 | Country bred | Contact D-68 | B.B. | B.B.,S.A. | Sterile | Sterile | Sterile | 17 |
| D-70 | | | B.B. | B.B. | Sterile | Sterile | Sterile | 17 |
| D-71 | | | B.B. | B.B. | B.B.,S.A. | S.A.,S.Y. | S.A. | 11 |
| D-72 | | None | Sterile | 1 colony S.A. | Sterile | Sterile | Sterile | 18 |
| D-73 | | | Sterile | Sterile | Sterile | Sterile | Sterile | 18 |
| D-74 | Pound | Pound | None made | B.B.,C.T. | None made | None made | None made | 16 |
| D-75 | Country bred | Contact D-74 | C.T. | C.T. | S.A.,H.S. | H.S. | C.T. | 19 |
| D-76 | | | C.T. | B.B.,C.T. | C.T. | C.T. | C.T. | 19 |
| D-77 | | | C.T. | C.T. | B.B. | S.A.,B.B. | C.T. | 19 |
| D-78 | | | C.T. | C.T. | Sterile | S.A.,C.T. | Sterile | 19 |
| D-79 | | | C.T. | C.T. | Sterile | S.A.,C.T. | Sterile | 19 |
| D-80 | | | B.B. | Sterile | S.A. | Sterile | Sterile | 5 |
| D-81 | | None | Sterile | Sterile | Sterile | Sterile | Sterile | 18 |
| D-82 | | | Sterile | Sterile | Sterile | Sterile | Sterile | 18 |



TABLE I—*Bacteriological findings in distemper dogs—Concluded.*

TABLE I—*Bacteriological findings in distemper dogs—Concluded.*

| DOG | SOURCE | SOURCE OF INFECTION | TRACHEA | LUNG | LIVER | SPLEEN | HEART BLOOD | REMARKS |
|---|---|---|---|---|---|---|---|---|
| D-83 | Country bred | Contact D-86 | B.B. | B.B. | S.A. | Sterile | Sterile | 11 |
| D-84 | | | B.B. | B.B. | S.A. | S.A. | Sterile | 11 |
| D-85 | | | H.S. | H.S. | Sterile | Sterile | Sterile | 11 |
| D-86 | Pound | Pound | B.B. | B.B. | S.A. | H.S. | Sterile | 12 |
| D-87 | Country bred | Contact D-86 | B.B. | B.B. | Sterile | S.A.,H.S. | S.A. | 11 |
| D-88 | | | B.B. | B.B.,S.A. | H.S. | Sterile | S.A. | 11 |
| D-89 | | | B.B. | B.B. | B.B. | B.B. | B.B. | 7 |
| D-90 | | | B.B.,C.T. | B.B. | Sterile | Sterile | Sterile | 12 |
| D-91 | | | Sterile | H.S. | Sterile | Sterile | Sterile | 7 |
| D-92 | Country bred | None | Sterile | Sterile | 1 colony S.A. | Sterile | Sterile | 18 |
| D-93 | | Indirect contact | Sterile | H.S. | Sterile | S.A. | H.S. | 5 |
| D-94 | | | S.A.,H.S., C.T. | S.A.,C.T. | Sterile | Sterile | Sterile | 19 |
| D-95 | | | H.S.,C.T. | H.S.,C.T. | Sterile | Sterile | H.S. | 20 |
| D-96 | Kennel | Contact in field trials | H.S.,S.A., C.T. | H.S.,C.T. | H.S. | H.S.,C.T. | H.S.,C.T. | 21 |
| D-97 | | | H.S.,S.A. | H.S.,S.A. | H.S. | Sterile | Sterile | 22 |
| D-98 | | | S.A. | S.B. | S.A. | Sterile | S.B. | 22 |
| D-99 | | | S.A.,H.S. | S.A. | H.S. | Sterile | S.B. | 22 |
| D-100 | Country bred | Indirect contact | S.A.,C.T. | B.B. | C.T. | H.S. | B.B.,C.T. | 22 |
| D-101 | | | B.B. | B.B. | S.A. | Sterile | Sterile | 23 |
| D-102 | Pound | Pound | B.B. | B.B. | Sterile | S.A. | Sterile | 6 |
| D-103 | | | B.B. | B.B.,S.A. | S.A. | Sterile | Sterile | 6 |
| D-104 | Country bred | Contact D-102 | S.A. | B.B.,C.T. | S.A. | S.A. | S.A. | 10 |
| D-105 | | Contact D-103 | S.A. | B.B. | S.A. | Sterile | S.A. | 7 |

been removed with sterile instruments. Five to 10 cc of blood from the heart was taken up in a sterile pipette and seeded in 250 cc of nutrient bouillon. When no growth occurred on the media thus seeded, the cultures were incubated for at least four days before being regarded as sterile. When growth occurred, individual colonies were fished and transferred to a suitable culture medium, after which the organisms were identified by the usual laboratory procedure.

After the cultures had been made from the various organs, the spleen, which Dunkin and Laidlaw[9] found to be exceptionally rich in filtrable virus, was removed with aseptic precautions, weighed, ground finely with sterile sand and made into a twenty per cent suspension in sterile physiological salt solution. This suspension, containing sterile glass beads, was then shaken in a mechanical shaker for thirty minutes, after which it was filtered through No. 8 Mandler filters. When known to be sterile, from tests on various kinds of culture media, 2 cc of this filtrate was injected subcutaneously into a normal ferret, the animal found by Dunkin and Laidlaw[9] to be extremely susceptible to the filtrable virus of dog distemper.

## Results of Bacteriological Studies

This report deals with only the bacteriological findings in these naturally infected cases and does not include the pathologi-

### Explanation of Table I

Abbreviations:—

B.B. = *Bacillus bronchisepticus.*
H.S. = *Hemolytic streptococcus.*
S.A. = *Staphylococcus albus.*
C.T. = Colon-typhoid organism.
S.Y. = *Staphylococcus aureus.*
S.L. = *Staphylococcus citreus.*
S.B. = Spore-bearing aerobe.

Remarks:

1. Destroyed when in early stages of distemper.
2. Ill 10 days when destroyed.
3. Advanced stage of distemper when destroyed.
4. Moribund when destroyed.
5. Ill 3 days.
6. Ill about 1 week.
7. Ill 4 days.
8. Contracted distemper 2 months after immunization with spleen vaccine and virus.
9. In advanced stages. Nervous symptoms. Brain sterile.
10. Ill 5 days.
11. Ill 1 day.
12. Ill 6 days.
13. Ill 3 weeks.
14. Ill 13 days.
15. Ill 8 days. Moribund when destroyed.
16. Cultures made after death from disease.
17. Ill 2 days.
18. Normal. Destroyed on arrival at laboratory.
19. Ill 6 days. Cultures after death from disease.
20. Ill 7 days. Cultures after death from disease.
21. Ill 5 days. Cultures after death from disease.
22. Ill 4 days. Cultures after death from disease.
23. Ill 36 days.

FIG. 1. Temperature chart of dog 101, an uncomplicated case of *Bacillus bronchisepticus* infection.

cal changes seen in the various animals examined nor the results of injection of ferrets with sterile filtrates of spleen suspensions following which the presence of a filtrable virus was not demonstrated.

For the sake of brevity, the bacteriological findings are summarized in table I. A study of this table indicates that *B. bronchisepticus* was by far the predominating organism found, having been isolated from one to five sources from 81 per cent of the cases studied. Next in order of their incidence are hemolytic streptococci (9 per cent), *Staphylococcus albus* (6 per cent), and organisms of the colon-typhoid group (4 per cent). (See table II.) *B. bronchisepticus* was found most frequently in the trachea and lungs, being present in these respective organs in 70 and 73 per cent of the cases. It was present also in the heart-blood in 15 per cent of the cases, and less frequently in the liver (6 per cent) and spleen (6 per cent). (See table III.) The hemolytic streptococci were often found associated with *B. bronchisepticus*, but in several instances this organism was apparently the causal agent. (See D-91, D-93, D-94, D-95, D-96, D-97, D-98, D-99, in table I). *Staph. albus* was frequently found associated with both of the above organisms but should be considered merely as a secondary invader, as should *Staph. aureus* and *Staph. citreus*. The presence of organisms of the colon-typhoid group in some of the cases was in all probability indicative of antemortem or postmortem invasion of these organisms from the intestinal tract since most of these animals had been destroyed when

TABLE II—*Predominating organisms in 100 naturally infected cases.*

| ORGANISM | BR. BRONCHISEPTICUS | STREPTOCOCCI | STAPH. ALBUS | COLON-TYPHOIDS |
|---|---|---|---|---|
| Number | 81 | 9 | 6 | 4 |

TABLE III—*Incidence of various organisms in internal organs of 100 cases.*

| ORGANISM | TRACHEA | LUNGS | LIVER | SPLEEN | HEART-BLOOD |
|---|---|---|---|---|---|
| *B. bronchisepticus* | 70 | 73 | 6 | 6 | 15 |
| *Staphylococcus albus* | 16 | 25 | 45 | 42 | 26 |
| Streptococcus | 8 | 10 | 10 | 8 | 5 |
| *Staphylococcus aureus* | 1 | 2 | 2 | 1 | 1 |
| *Staphylococcus citreus* | 0 | 3 | 1 | 1 | 0 |
| Colon-typhoids | 17 | 14 | 3 | 6 | 6 |
| Sterile | 5 | 4 | 32 | 44 | 44 |

moribund or had died from the effects of the disease before the cultures were made.

The incidence of *B. bronchisepticus* in the cases studied is comparable to the findings of Torrey and Rahe[5] (81 per cent) and of Schoiche[13] (85 per cent), but is slightly less than those of M'Gowan[4] (93 per cent) and of Ferry,[3] who found the organism in all of the cases reported. Hardenbergh[7] isolated this bacillus from 66 per cent of his cases. This variation in incidence of *B. bronchisepticus*, as reported by these authors, may possibly have been due to differences in methods or to the condition of the dogs, *i. e.* the length of time which had elapsed from the onset of the disease and the time they were autopsied for bacteriological examination.

A number of investigators consider *B. bronchisepticus* as a secondary invader only, which, under ordinary conditions, may be present in the animal body without having any pathogenic action until the animal becomes infected with the filtrable virus of dog distemper. Then the resistance is lowered sufficiently for the bacillus to become pathogenic and produce the symptoms that arise during the progress of the disease. Undoubtedly, certain animals may be carriers of *B. bronchisepticus*, as was shown by Ferry[11] and also by the author,[12] who found this organism to persist in the lower trachea some three weeks after the disappearance of the symptoms of distemper. On the other hand, this organism has been shown to be the primary invader in the symptom complex commonly diagnosed as canine distemper by Ferry,[2,3] M'Gowan,[4] Torrey and Rahe[5] and Schoiche,[13] as well as being responsible for primary infections in guinea pigs, [4,11,14] rabbits[4,11,15,20] ferrets,[4] cats,[4] white rats, [4,21] monkeys, [4,11] and also in conditions in human beings which simulated whooping-cough[20] and the common cold.[22] Batt[23] has recently reported an outbreak of a contagious disease of dogs in which *B. bronchisepticus* was the etiological factor.

Further evidence that *B. bronchisepticus* is a distinct pathogen, capable of producing the symptoms from which a diagnosis of canine distemper is usually made, is shown by the fact that the organism can usually be very readily recovered from pups reared in localities free from infection and later exposed to distemper by contact with a naturally infected animal. (See D-23, D-24, D-56, D-57, D-58, D-59, D-70, D-71, D-87, D-88, D-89 and D-90 in table I.) Pups so reared usually develop symptoms of distemper in seven to nine days after contact with the infected

animal. Usually no difficulty was experienced in the isolation of *B. bronchisepticus* when the pups were destroyed and cultures made on the day of the appearance of the first symptoms, *i. e.* cough, serous or mucous nasal discharge and elevation of temperature, which usually occurred simultaneously with the first-mentioned symptoms. In some few instances, however, even though the clinical symptoms were similar, *B. bronchisepticus* was not recovered but a hemolytic streptococcus was apparently the causative factor. (See D-85, D-91 and D-95, in table I.)

In contrast to these findings, cultures made from the internal organs of litter-mates of puppies used in the above experiments were almost invariably sterile if made when the animal was destroyed before any exposure to disease had taken place. (See D-72, D-73, D-81, D-82 and D-92 in table I.) The one colony of *Staph. albus* found on the plate made from the lung of D-72 and on that from the liver of D-92 may possibly have been a contamination.

To obtain further evidence of the pathogenicity of *B. bronchisepticus* and its ability to produce the symptom complex ordinarily diagnosed as canine distemper, several susceptible pups (D-61, D-64 and D-65) were infected by intratracheal injections of the organisms. Since it is a well known fact that this organism has a tendency to lose its virulence when carried for some time on artificial culture media, the strain used for these experiments was one isolated about two weeks previously from a naturally infected case (D-47) and had been transferred but once during that time. The growth from this transplant was suspended in physiological salt solution and diluted to a density comparable to 2000 million *Bacillus typhosus* per cc. One cc of this suspension was injected directly into the trachea by inserting the needle of the syringe between the cartilaginous rings. All pups developed typical symptoms of distemper and on autopsy no difficulty was experienced in recovering the organism.

## Summary

The results of studies of one hundred dogs showing the symptom complex commonly diagnosed as canine distemper showed that *Bacillus bronchisepticus* was present in 81 per cent of the cases.

A hemolytic streptococcus was found in 9 per cent of the cases examined.

In a still smaller group (6 per cent) *Staphylococcus albus* only was recovered. This organism was not thought to have been a primary etiological factor but a secondary invader.

In a very few cases, organisms of the colon-typhoid group predominated. These may possibly have been primary causative factors but it would seem more logical to believe that they were either antemortem or postmortem invaders, since most of the animals from which these were recovered were either moribund at the time they were destroyed for culturing or had died from the effects of disease before bacteriological examinations were made.

Susceptible pups, which had been reared in localities free from infection, were seen to develop symptoms of the disease clinically diagnosed as canine distemper, in from seven to nine days after exposure to infection by contact with a naturally infected dog. From the majority of these animals, no difficulty was experienced in isolating *B. bronchisepticus*, on the day the first symptoms were noted. In a few cases a hemolytic streptococcus was found. These findings were in contrast to negative bacteriological results obtained when cultures were made from the internal organs of susceptible pups destroyed before exposure to any infection.

The symptom complex of canine distemper was produced in susceptible pups following the intratracheal injection of suspensions of recently isolated *B. bronchisepticus*.

## Conclusions

Since *Bacillus bronchisepticus* was the predominating organism found in 81 per cent of the naturally infected cases studied, and since it was recovered from the majority of the susceptible animals after development of symptoms of distemper following contact with naturally infected dogs, and since these same symptoms were produced in other susceptible dogs by intratracheal injections of suspensions of recently isolated cultures, this organism should be regarded as a primary etiological factor in the symptom complex clinically diagnosed as canine distemper.

Hemolytic streptococci are probable causative factors in some cases showing similar symptoms.

*Staphylococcus albus* should be regarded as not having primary etiological importance in this disease but as a secondary invader.

The organisms of the colon-typhoid group found in a very few of the cases studied should probably be regarded as being

either antemortem or postmortem invaders rather than as primary causative factors in the disease although there is the possibility that they may be primary factors in the so-called "intestinal" form of canine distemper.


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## DISCUSSION

DR. ADOLPH EICHHORN: To some extent, Dr. Schlingman's report is more or less a repetition of previous investigations which have been made to establish that the *Bacillus bronchisepticus* is the etiological factor of canine distemper. Some of the investigators who have studied the bacteriology of canine distemper have definitely established the presence of the *Bacillus bronchisepticus* in a great number of cases and I do not doubt that Dr. Schlingman has isolated the organism in 80 per cent of cases.

Nevertheless, it is still my opinion—based on my own experiments and on those conducted in England—that the *Bacillus bronchisepticus*, even when present, is not the primary etiological factor. It is possible that Dr. Schlingman conducted his bacteriological work at the time when the virus had already vanished from the blood.

We know that in many diseases caused by filtrable viruses the virus is present only in certain stages of the disease. Thus, for instance, in hog cholera it is present only in the acute stages. In canine distemper, it is possible that other organisms, as the *Bacillus bronchisepticus*, may be present in the animal even simultaneously with the virus and still remain there after the virus has disappeared. Therefore, the isolation of the *Bacillus bronchisepticus* does not by any means exclude the possibility of the presence of the virus.

From the extensive studies which have been conducted and also from personal observations, there should be no longer any doubt with regard to

the primary factor of canine distemper and it should be considered an established fact that the disease is caused by a filtrable virus.

In our establishment, we have inoculated approximately 5000 puppies. In this work, we have found that a certain number, about 20 to 30 per cent, are not susceptible, but in all susceptible animals we have observed the development of classical distemper, such as elevation of temperature, mucous discharge from the eyes and nose, with which gastro-intestinal symptoms or nervous manifestations later became associated. If these animals were destroyed and ferrets inoculated with a small portion of the spleen, we never had any difficulty in transmitting the disease to at least 98 per cent of the ferrets. It should be understood that these ferrets were at all times strictly isolated from any possible contact with canine distemper. Such has been our experience on several thousand ferrets and the same experience has been obtained in England also. Thus, it is evident that we can no longer doubt that a filtrable virus is the cause of true canine distemper. We should, however, realize that not all cases diagnosed as canine distemper are really true cases of virus distemper, and it is realized that the accurate diagnosis of canine distemper from clinical manifestations is not simple and in many cases the diagnosis of canine distemper is made when some other conditions might be responsible for the symptoms in the animals.

As to the relation of the *Bacillus bronchisepticus* to canine distemper, I personally conducted some investigations along this line at the time I was associated with the Bureau of Animal Industry, in 1912 and 1913. I isolated the bronchisepticus organism from many cases of so-called distemper and many of these, no doubt, were true distemper cases, but I have never succeeded in transmitting the disease to dogs of all ages by inoculation of pure cultures of these organisms.

Considering also the fact that aside from bronchisepticus, other organisms are found in cases of distemper, we must accept them as secondary invaders, because neither through inoculation nor contact was it possible to transmit canine distemper with any of these organisms.

The complement-fixation test which has recently been applied in England for the evaluation of the hyperimmune serum and which is based on the filtrable virus being the causative factor of the disease must also be accepted as further evidence that the virus is the cause of the disease. It has been found that the spleens of ferrets dead with the filtrable virus of distemper are an effective antigen when employed for the evaluation of sera from hyperimmunized dogs. I believe that the possibility of the application of this biological test, and the proof of the antigenic value of the tissue containing the virus, together with the numerous other definite evidences produced, should no longer create a doubt in anyone's mind as to the cause of canine distemper.

DR. J. W. BENNER: I wonder, in those intratracheal injections which Dr. Schlingman made, if there was not a possibility of a small, very small, quantity of filtrable virus being carried over in those cultures of bronchisepticus. They were, as I understand it, cultures of organisms recently isolated from cases.

DR. SCHLINGMAN: Carried through only about one transfer, there might be a possibility, but I rather doubt it, simply because these cultures, after isolation and incubation at 23° C., were transferred at once and the slants were held two weeks after isolation. According to the early work of Laidlaw and Dunkin it appeared that the filtrable virus would die out in that two weeks of high temperature.

DR. BENNER: The same cultures were not subjected to injection into other dogs?

DR. SCHLINGMAN: It is very difficult to produce the disease by subcutaneous injections.

DR. BENNER: I wish to take exception to the statement which Dr. Eichhorn made, in which he said that filtrable virus is present in the blood only in the very acute stages. It seems to me that the filtrable virus is present throughout the moribund stage, and even after death. Hog cholera seems to be different from dog distemper. In dog distemper there seems to be a stage,

and it is a very acute stage, where this virus is present in the blood, but in hog cholera, as far as I have been able to find, the virus is present in the blood throughout the entire stage of the disease.

MAJOR R. A. KELSER: I suppose a number of you are familiar with the work of Hadley, at Ann Arbor, with bacteria, in which he has found filtrable stages of organisms which have been considered as the primary cause of diseases without any such thing as a filtrable stage. The thought has arisen in the minds of those who have followed this work of Hadley that probably some of these organisms, over which we have had some dissension as to etiological significance, may have a filtrable stage. He has found filtrable stages for a number of the organisms of the paratyphoid group, so that it is possible that further work may show that some organisms, ordinarily considered secondary factors, as *B. bronchisepticus*, in the case of canine distemper, and *B. suipestifer*, the old hog cholera organism, may be bacillary forms of the bacteria having filtrable stages, the bacillary forms not being capable, ordinarily, of producing primary disease, but the filtrable stages having this power. It is possible that subsequent work may indicate that there is some connection of this sort—the so-called filtrable virus, capable of producing the disease, may be a stage in the life cycle of virus capable of producing a bacillary form.

DR. EICHHORN: I hope that my statement has not been misunderstood. I have aimed to imply that in certain stages of canine distemper it might be difficult or impossible to demonstrate the filtrable virus because, from experience with other diseases, we know that the filtrable virus is present in the most concentrated form only in certain stages of the disease, especially during the early febrile period, and later it may diminish and finally disappear, and the animal might then become the prey of the pathogenic action of the secondary invaders.

## Comments on the Directory

"The new membership directory was received several day ago, and I think that it is a very creditable piece of work."

—Dr. J. P. Foster, Minneapolis, Minn.

"Permit me to extend my congratulations on the very fine directory of the A. V. M. A., which you have just gotten out."

—Dr. C. M. Haring, Berkeley, Calif.

"The directory is certainly a credit to the work of the Secretary's office."

—Dr. Wm. Herbert Lowe, Paterson, N. J.

"I wish to congratulate you on the good directory of the A. V. M. A."

—Dr. C. J. Marshall, Philadelphia, Pa.

## Missouri Valley Veterinary Association Votes on Disbanding

The Executive Board of the Missouri Valley Veterinary Association has authorized Dr. E. R. Steel, Secretary-Treasurer, to secure a post-card vote of the members in good standing on the question as to whether or not the organization should disband. In the notice mailed to members, it is stated that present conditions make it difficult to maintain the membership and secure attendance at meetings.

# MENINGO-CEREBRAL COMPLICATIONS IN CANINE DISTEMPER*

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In discussing the above subject it is necessary first to take into consideration some generalities and specific facts concerning true canine distemper in its uncomplicated form.

Carré,[1] in 1905, announced the results of his work showing that canine distemper is caused by an ultramicroscopic organism, or a filtrable virus. In 1911, Ferry,[2] M'Gowan[3] and Torrey and Rahe[4] working independently, apparently demonstrated that the *Bacillus bronchisepticus* is the cause of canine distemper. In 1925, Lockhart and associates[5] published the result of their work by which it was shown that a true virucidal serum could be prepared in dogs hyperimmunized against the filtrable virus of Carré. In 1926 and subsequently, Dunkin and Laidlaw[6] published reports showing the correctness of Carré's findings. Therefore, it is now generally conceded that the cause of true canine distemper is the filtrable virus of Carré.

In 1926, one of us (A. L.)[7] read a report before this Section dealing with the development of canine distemper. A few months later, Dunkin and Laidlaw[8] published a report corroborating the above in all essentials. It is shown by these reports that true distemper, when it remains uncomplicated, is an acute febrile disease characterized by a peculiar double temperature curve, marked debility, inappetence and, in those cases which recover, running a course of from three to five weeks. These reports have clearly shown that in young dogs known to be free from distemper, until definitely exposed to distemper, complications do not develop until the dog has been abnormal for a considerable period of time. In no case have we observed purulent nasal discharge, pneumonia, dysentery or nervous manifestations until several days after the secondary rise of temperature.

Having observed the regularity of the development of filtrable virus distemper and the comparative regularity of the development of those symptoms and lesions which are usually classed as certain types of distemper, we were led to undertake a study of

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the various types of distemper, with a view to determining their true nature. In the course of this study we became convinced that the so-called types of distemper are dependent upon secondary infection and in reality are complications of the true filtrable virus disease. It has also been our observation that under certain conditions these manifestations, which are ordinarily secondary, are primary, and may occur without the assistance of the filtrable virus of Carré.

Of the various complications of canine distemper, it is perhaps true that the so-called nervous manifestations are the most important, especially from the standpoint of prognosis. In some sections, as high as 50 per cent of all cases of canine distemper develop nervous manifestations and frequently 75 per cent or more of such cases prove fatal. It is not necessary to describe this complication to any group of veterinarians engaged in small-animal practice. But, in order that we may avoid confusion, we shall consider only that type of nervous derangement which develops during, or immediately following, an attack of true distemper.

It should be borne in mind, of course, that there are many things which may produce spasms in dogs, and which are sometimes difficult to differentiate from the nervous complications occurring in distemper. Parasitism, chemical action of certain drugs, disturbed metabolism, foreign bodies in the stomach or intestine and perhaps other factors occasionally complicate diagnosis; but, as a general rule, the type of nervous manifestations occurring as a complication of distemper is very characteristic and should not be confused with spasms originating from other causes. In exposing to distemper several thousand healthy young dogs obtained from rural districts and known to be free from disease, we have never observed the development of so-called distemper spasms until at least twenty-one days had elasped after definite exposure to distemper.

In dogs which have died as a result of this complication and in those destroyed during its advanced stages, the pathological changes are fairly uniform and characteristic. The meninges of the brain are usually congested, somewhat thickened and rather cloudy in appearance. The brain proper shows little macroscopic change. The ventricles of the brain contain an abnormally large amount of fluid, which is usually somewhat opaque and contains small flocculi. The heart frequently contains endocardial hemorrhages and sometimes epicardial hemor-

rhages are found. The spleen is usually enlarged and presents some subserous hemorrhages.

Rosenow[9] has described a condition the result, of an experimental inoculation, which is very similar to, if not identical with, the condition under discussion. To quote from Rosenow:

> The microscopic picture, although highly characteristic, varies considerably, depending on the duration and severity of the symptoms. In the animals that die relatively soon of especially severe symptoms, meningeal infiltration by polymorphonuclear leukocytes and round cells, with similar but less marked localized parenchymatous and perivascular infiltration, dominates the picture. Polymorphonuclear leukocytes may be as numerous as round cells, especially in the meninges. Localized hemorrhages in the pia and substance of the brain, especially in the basal ganglions, are common. In animals that live for a longer time, the proportion of mononuclear cells in the meninges and perivascular spaces is greater, and meningitis is less pronounced and is limited to the perivascular spaces.

In our experience we have sometimes found, on microscopic examination, some degeneration of nerve cells, particularly of the motor type nerve cells, with the occurrence of bodies which might be confused with Negri bodies. These pseudo-Negri bodies may be differentiated from true Negri bodies by their indistinctness of outline and their freedom from nuclear-staining granules. Stained preparations of fluid from the lateral ventricles and from impressions made from the pia mater quite constantly reveal the presence of peculiar bacterial organisms. In so far as we are aware, the first reference to this organism was contained in the report of Lockhart, made in 1926 before this Section, when the organism was described as belonging either to the class of diplococci or streptococci.

## Bacteriology

In view of constantly finding the organism referred to above, in cases of distemper spasms, we have endeavored to determine whether or not it is the cause of this condition. One of us (S. R. J.), working independently from 1923 to 1927, observed, isolated and worked with the same organism, occurring probably as the cause of a primary disease of foxes. We have been able to recover this organism from the blood stream of affected animals in a large majority of those animals studied, and it has been recovered from the brain and lateral ventricles of almost all animals. When injected into suitable animals, under proper conditions, it is capable of producing typical symptoms and lesions of so-called distemper spasms.

When first isolated, the organism usually produces a small, slightly elevated, moist colony. On blood plates the colony is

surrounded by a narrow greenish zone and in some cultures by a narrow zone of hemolysis. A few cultures, when isolated, do not possess the ability to produce the green or hemolytic zone on blood plates, and cultures which have lost their virulence may also apparently lose this characteristic. Glucose broth is rendered turbid and after 18 to 24 hours becomes granular, followed by settling out after 24 to 48 hours. Growth occurs in plain broth but is very slight and slow. Acid is produced in glucose, lactose, saccharose and levulose. No acid is formed in mannitol. Acid is occasionally formed in raffinose. No gas is formed in any of the above sugars. Litmus milk is decolorized, followed by some digestion.

In young cultures from blood agar or glucose broth, the organism is Gram-positive and frequently pleomorphic. The size and shape of the cells ranges from regular cocci, arranged in formation similar to staphylococci, to diplococci or short-chained streptococci, or lanceolate diplococci. The size of the individual cells varies from a very small micrococcus to a large cell approximating the size of a yeast cell. In some old cultures all sizes and arrangements may be found in the same field.

Rosenow[9] reports his work with an organism apparently exactly corresponding to this, which he has isolated from epizoötic encephalitis of the fox and which he has shown to be pathogenic for dogs when injected into the eye or subdural spaces. It is interesting to note that Rosenow has isolated this organism also from supposedly sterile filtrates of fox brains submitted to him by Green and associates.

Schlingman[10] has discussed the possibility of a streptococcus being associated with canine distemper.

## Immunization

In June, 1929, we undertook the development of an immunizing agent to control this complication of canine distemper. A bacterin was prepared from these pleomorphic streptococci and it has been regularly used in our establishment since that time. Before this bacterin was used we experienced considerable difficulty as a result of injecting dogs with blood containing this organism. Since making use of this bacterin as a routine procedure, exceedingly few cases of spasms have occurred in our establishment. We have injected immunized dogs with blood known to contain large numbers of these streptococci without producing symptoms of this infection. A number of veterinarians

in various parts of the United States have coöperated with us in testing the efficiency of this bacterin in preventing the development of distemper spasms. Reports from these practitioners are incomplete, but in every case where a report has been rendered it has been said that the use of this bacterin has resulted in materially decreasing the frequency and severity of the development of nervous complications of distemper.

Rosenow has shown that by the injection, into the cisterna magna, of neutralized cultures of the streptococcus with which he was dealing, solid immunity against infection could be produced. It therefore seems that immunization against this type of infection may be expected to be reasonably successful.

## Treatment

Medicinal and biological treatment of the nervous manifestations of distemper has always been considered unsatisfactory. We attempted to develop an immune serum for this purpose, but met with only partial success. Many of the practitioners who have coöperated with us have informed us that where this bacterin was used, soon after the onset of symptoms, recoveries resulted in more than half the cases. In view of Rosenow's work in injecting bacterin into the cisterna magna, it is possible that if concentrated immune serum was injected in the same location, it would produce desirable results as a curative agent.

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## Discussion

Dr. K. U. Jones: In using this bacterin in established cases of distemper, do you really get immunity against this infection in the meninges at the same time?

Dr. Lockhart: I don't know whether I understand the question very well. The method that most of the practitioners have followed is to take their cases of distemper that have not yet shown the nervous manifestations and use this

bacterin along with the regular distemper treatment they ordinarily use, and attempt to prevent the development of these nervous manifestations. That is our method with dogs we are putting through canine distemper. We put a number of them through intentionally. We repeat the dosage every five days.

DR. O. B. MORGAN: In discussing Dr. Green's work at the University of Minnesota—I would like to ask Dr. Lockhart if Dr. Green thinks at the present time there is such a thing as distemper of foxes that affects canines and if this encephalitis he is dealing with is merely the secondary infection.

DR. LOCKHART: I could not say definitely what Dr. Green might think. His contention is that encephalitis itself is a specific disease caused by a filtrable virus.

DR. C. F. SCHLOTTHAUER: I have never had anything to do with Dr. Rosenow's work but I remember when Dr. Green was down there working with Dr. Rosenow, he apparently infected dogs and foxes with the same virus.

I want to mention this—I put Dr. Lockhart on the program for the reason that he is the first man in the world, as far as I know, who made a useful anti-distemper serum. We sometimes forget that. Because a man is our neighbor we are prone to overlook, sometimes, the great things that he does. I think Dr. Lockhart has done a very wonderful thing. All of us who use much canine distemper serum and use it logically and in a rational way, have to report good results.

When we get puppies in and want to avoid distemper, I have never yet seen one develop distemper if it had been given a sufficient dose of serum and put in a pen where we knew distemper infection was present. Ten cubic centimeters in a half-grown fox terrier puppy is not always sufficient. I have found twenty cubic centimeters to be sufficient, and repeat the dose in four or five days.

It may be pure luck, or we may not have a virulent form of distemper in our kennels, but I think we probably have the same type every one else has. I have purchased some valuable puppies for members of the staff, everything from Boston terriers, English bulls, Chows, Scotch terriers, and so on, and I have such confidence in the anti-serum that I tell them without reservation to go ahead and buy their dogs and that If they watch them closely there is no reason to lose them from distemper. I might change my mind in the future but this is the experience of five years.

DR. D. A. EASTMAN: Dr. Wright and myself have been using the bacterin Dr. Lockhart speaks about, not with any laboratory efficiency as to records, but for a time we practiced giving it to every other case of distemper and I am sure the nervous symptoms developed much less in those we gave the bacterin than in those we did not. I believe it has some value.

DR. C. W. BOWER: Relative to the bacterin, I have used it, just as Dr. Eastman and the others have. I cannot say what percentage of success we had in preventing the condition but I should say perhaps sixty per cent. I gave them the bacterin immediately, when the first symptoms of distemper became noticeable. I also attempted to use it as a curative, without any results, as far as I could tell. I didn't expect any, but that is my report—no results secured.

DR. LOCKHART: There is one thing I should like to emphasize about this—the object in presenting this subject was to attempt to show that these nervous manifestations are not in reality entirely dependent upon the filtrable virus of distemper. I am of the opinion that in most cases it is necessary for something to pave the way for this type of infection and that the filtrable virus of distemper is the usual thing which paves the way for it.

I may say further concerning the treatment of the disease, having examined a good many brains from infected dogs, it is rather difficult for me to see how anything is going to be of much benefit in treating these dogs. There is considerable degeneratiln of nerve cells, and I don't see how any agent will ever be developed that will do a great deal of good in cases that have already developed nervous manifestations.

This bacterin is still purely an experimental product and is not being offered for sale.

# TREATMENT OF THE DISEASES OF THE EYE AND ACCESSORY ORGANS*

*By* D. A. EASTMAN, *Moline, Ill.*

In discussing the treatment of diseases of the eye and its accessory organs, I will try to confine this paper to a discussion of those diseases which are most numerous and, therefore, most important. These conditions and their treatment are not new and no doubt are not especially different from what most of you are using, but a review of those conditions which we see every day will perhaps be of some benefit.

The treatment of the diseases of the eye, in the dog, must take into consideration the fact that the eye of the dog has two very distinct functions. The first is beauty and the second is efficiency. It is my opinion that the former is more often more important than the latter. I do not believe that this situation is true in any other animal or in the human.

The eyes of dogs vary so much anatomically with the various breeds, and in breeding toward the standard of any given breed, the attempt to produce eyes of a given character leads often in turn to many diseased conditions and abnormalities. Size, shape, color and expression of the eye become very integral parts of the conformation of the head of the breeds and in many cases these characteristics, through selective breeding, are carried to the extreme, thereby predisposing to diseases not to be found in a species of animals whose eyes are more or less uniform.

It then becomes our duty, in the treatment of diseases of the eye, to take into consideration all these differences of appearance and to direct any treatment of them toward the reduction of the abnormal appearance of the eye in the particular breed with which we are working and, in some cases, even going as far as to produce a normal appearance in an eye which originally was not normal. At the same time, this wide variation in the conformation of the eye is directly responsible for many eye conditions that not only lessen beauty but damage efficiency.

Also, many types of eyes apparently are not highly efficient as organs of vision, due principally to the man-made characteristics which have been brought into these eyes by selective

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breeding. As an example of this and taking the two extremes, we could mention the Pekingese and the Chow. In both, the eyes are all out of proportion to the conformation of the rest of the body. The small squinty eye of the Chow predisposes, far more than any other breed, to entropium; the bulging, prominent eye of the Pekingese to corneal injury and ulcer as well as luxation. The sagging lids of the hounds and setters are excellent receptacles for foreign bodies.

In all of these, as well as many more, the normal formation and appearance of the eye and its accessory organs are responsible for many pathological conditions. Therefore, we are not so much concerned with the efficiency of the eye as an organ of vision and are not particularly interested in problems of correct vision nearly so much as we are interested in problems of correct structure, deviations from it, and diseases resulting therefrom.

### Enlargement of Harder's Gland

Starting then with one of the commonest and simplest conditions which we meet and which is in all probability due to unusual eye formation, we will discuss the condition which is known to the layman as "haw." It consists in the enlargement of the gland of Harder, which is a normal structure attached to the inner surface of the membrana nictitans. Normally it is not visible and its size is of no importance. However, upon enlargement, it may constitute an abnormality even without being visible above the edge of the membrane, although, usually, it is. It is found especially in dogs with rather prominent eyes, particularly in Bostons and beagles.

In the majority of cases, the enlargement appears as a round, reddish mass, the size of a pea, lying in the inner canthus of the eye. It does no damage to vision as a rule, and in most cases is not unduly uncomfortable, its only undesirable effect being its looks. Often it does not protrude above the edge of the membrane but, because of its enlargement, forces them embrane upward, even to the point of covering a portion of the cornea. Its removal is indicated in either case and is the only effective treatment. The technic of its removal is known to you all, merely consisting of grasping it with forceps and clipping it off at its base with scissors. However, there are some points about it that might bear discussion.

The first is the anesthetic to be used. I prefer, above all others, cocain in a 1 per cent solution, for two reasons. First,

the anesthesia is more profound and more quickly established. More important, it also acts as a hemostatic. The use of any of the other locals also necessitates the use of adrenalin to control what may be very stubborn hemorrhage. The hemorrhage following the use of cocain is very slight and of no importance at all, pressure exerted to the eye for a minute or two following the operation being all that is necessary to control it. After-treatment should consist of the application twice daily of argyrol, neo-silvol, or any other accepted eye antiseptic.

Another important thing, I believe, is the fact that we do not remove them in as many cases as we should, too many times confining their removal to the visible ones. There are many eyes, and especially is this true in Bostons, the appearance of which can be greatly enhanced by the removal of this gland from its normal position behind the membrane, thereby allowing the membrane to fall or rather shrink into the inner canthus, producing in appearance a much larger, clearer and cleaner eye, a thing which is much to be desired in all dogs—and especially good Bostons. The average owner usually does not suspect the cause of the condition or realize that an improvement can be made so simply and it is it our duty in many cases to inform him regarding it.

Actual removal of a portion of the membrana nictitans is occasionally necessary, using approximately the same technic as for removal of the "haw," but it is surprising the number of instances in which this is not at all necessary if the removal of the gland, even though it is not visible, is practiced.

### Entropium

Entropium, the rolling in of the edge of the lower lid, is perhaps our next commonest of the minor diseases of the accessory organs of the eye. As mentioned previously, it is so commonly seen in Chows that many breeders actually believe it is normal. It is one of the most painful, as well as unsightly, conditions that we meet. A single hair, even of minute size, in our own eye, drives us nearly frantic, yet hundreds of Chows, as well as dogs of other breeds, are allowed to live for months and years with entire outer surface of the lower lid continually scratching against the eye-ball. The eyes are squinted, many times almost entirely shut and they discharge continuously.

Again, a discussion of the technic of the operation is not so important, being well understood, as it is to urge early diagnosis

and prompt treatment from a humane standpoint as well as increasing both the beauty and efficiency of the eyes. The operation, as you know, consists in the removal of an elliptical piece of skin from the lower lid, the long axis of the incision being parallel to the edge of the lid. It is desirable to remove only the minimum amount of skin, which will suffice, when the opening is sutured, to shorten the lid sufficiently to roll it outward to its normal position. However, considerable leeway, one way or the other, can be given and still the lid, when healing is complete, will revert to its correct position.

The ordinary surgical proceedures of cleanliness and antisepsis are, of course, well observed. It is my experience that the anesthetic, whenever possible, should be local or at least a combination of morphin and a local anesthetic. I say this because I believe the lid should always be operated with the dog in his normal standing or sitting position and with the eye-lids not completely relaxed. Otherwise, it is hard to determine correctly the proper location of the incision and the proper amount of tissue to be removed.

### Butyn Preferred for Anesthesia

As a local, I prefer butyn in a 2 per cent solution, injected in minute quantities in a circle around the line of incision. It gives complete and lasting anesthesia and does not delay healing to any great extent. If the dog is tractable, that should be sufficient. If not, one-fourth to one-half grain of morphin aids the situation immensely and yet does not completely relax the patient. An after-dressing of any soothing ointment (I personally prefer pellitol) aids greatly in relieving the itching incident to healing and prevents the scratching or rubbing out of sutures. Suturing, incidentally, should be of silk, and interrupted, to prevent any possible longitudinal puckering of the wound.

Wounds and ulcers of the cornea are so frequent that any discussion of them seems almost superficial. However, the recommended lines of treatment of them are so varied that perhaps they are worthy of some time. There is just one thing I would like to emphasize in addition to any other treatment which may be used and that is the relief of pain. To me that is more important than all of the other treatments combined. The pain incident to any acute corneal disease is intense, causing a continual winking of the lids, rubbing back and forth over the injured or inflamed area, to say nothing of the fact that many cases continue to rub them with their paws.

The rapid and easy healing of the cornea cannot possibly be accomplished unless this irritation is relieved and it can be relieved only by the reduction of pain. Mechanical appliances for this purpose are worthless. I believe that, in by far the large majority of cases of both corneal ulcer and corneal injury, barring, of course, the removal of foreign bodies, treatment towards the relief of pain is all that is necessary.

I doubt very much the wisdom of the use of any severe antiseptic or any other method of treatment which is at all radical. Cocain solution (1 per cent), used in the eye as often as is necessary to maintain comfort, is I believe, the best single treatment we can use. There are also eye ointments on the market which accomplish the purpose of the relief of pain quite efficiently. The best of these that I have found is known as quinocaine ointment. It is both anesthetic and antiseptic, producing quite prolonged, mild anesthesia and is incorporated in a lanolin base which permits easy, even application. It is especially valuable as a dispensing agent, in that the owner can apply it in the eye much easier than he can a watery solution. The avoidance of exposure to light either by placing in a darkened compartment or by bandaging of the eye, when this is practical, is also a tremendous aid in the relief of pain. Most corneas will do a marvelous job of healing if allowed to heal without interference of the lids or feet. The hemostatic properties of cocain are also of value in controlling the hyperemia which is always present in the sclera in this condition.

### Rupture of the Cornea

When rupture of the cornea occurs, following either injury or ulcer, the application of a pressure bandage, together with the agents just mentioned for the relief of pain, constitutes what is usually the only necessary treatment. I prefer a many-tailed bandage of muslin with holes cut for the ears and the normal eye, and fastened under the throat with a pack of sterile gauze over the affected eye. Even though the anterior chamber may collapse due to rupture, continuously maintained anesthesia and pressure will usually suffice to attain prompt and correct healing, allowing the anterior chamber to refill, leaving the eye still usable and not particularly unsightly except for the presence of scar tissue. Incidentally, the amount of scar tissue is tremendously cut down if healing occurs without irritation, the same rule applying in that respect to wounds of the cornea that

would to wounds of the skin. The eye, from the standpoint of ability to recover from even tremendous injury and disease and still be a usable organ, is not at all delicate, as is so commonly believed by many.

As an antiseptic in these cases, when any is considered necessary, I believe that mercurochrome in ointment, preferably lanolin, is as good as any that we have.

### Inflammation of the Lids

The various degrees of inflammation of the lids are caused by many things, and are possibly the next most common condition that we meet with, and, while not at all serious in themselves, still are often very troublesome to treat, due to the fact that they are very irritating to the dog, and consequently he does everything that he can to delay healing of them through rubbing. This inflammation may vary from a slight superficial dermatitis to a generalized inflammation of the entire structure of the lids, resulting in extreme swelling, redness and pain and, later, following further injury to the lid, baldness and weeping.

Aside from removal of any direct cause which may be present, the generalized treatment that we have found most efficient is either the continuous application of hot, moist dressings, or, perhaps better still, antiphlogistine packs which are changed as frequently as they dry out. The results from the latter, both from the standpoint of comfort to the dog and rapid reduction in size of the swelling, with consequent healing, are very gratifying. The pack is held in place with the usual many-tailed bandage, except in some of the short-nosed breeds, in which this type of bandage is not permissible. In these latter, however, it is usually possible to hold the pack in place with tape ,changing the tape each time the pack is changed and thereby avoiding any cutting or irritation to the skin by it.

It is not uncommon for abscesses to develop within the lids following severe bruises and during the course of inflammation from any source. Prompt draining of them, of course, is essential to rapid healing. No irritant antiseptic should be used in the after-care of these abscesses, due to the possibility of their gaining entrance to the conjunctiva. In those cases of milder inflammation, especially accompanied by raw skin surface, butesin picrate ointment is applied and often does away with the need of bandaging, being both antiseptic and locally anesthetic in its action.

Glaucoma, or hydrophthalmus, is a condition seen often enough to justify some discussion concerning its treatment. In my experience, complete recovery from this condition can be attained only in its milder forms or where treatment is instituted very early. It, of course, is a common sequence of many other eye conditions, especially corneal diseases, as well as appearing apparently spontaneously. In the milder stages of it, pressure bandages applied over the eye apparently are of some value, at least in preventing further filling of the anterior chamber. However, where there is extreme enlargement of the entire eye-ball, with protrusion through the lids to the extent that closure of the lids is impossible, puncture of the cornea and draining of the anterior chamber is indicated. This should be done with a very fine cataract knife or the discission needle used in cataract operations.

The opening, through the cornea, should be as small as will possibly allow the escape of the fluid. Immediately following the escape of a portion of the fluid, allowing the eye to revert to the approximate normal size, a pressure bandage is applied to prevent, as much as possible, the further escape of fluid and permit healing of the wound. It also serves, if sufficient pressure can be exerted, to prevent further refilling of the anterior chamber. Movement of the iris should be maintained by the daily instillation of atropin into the conjunctiva. Also, it is well to carry on the ordinary treatment for keratitis which, as has been mentioned, consists chiefly of the agents aimed at the reduction of pain.

Hemophthalmus may occur as an acute stage of glaucoma or it may be caused by traumatic injury to the eye. Practically the same rules for treatment apply as in glaucoma. However, clots of blood may remain in the anterior chamber for some time but will usually be resorbed.

### Prolapse of the Eye-ball

Prolapse of the eye-ball is extremely common and the cause, as you know, is nearly always traumatic, the rubber tires of automobiles being the chief offenders. The treatment varies, both with the severity of the prolapse and the length of time elapsing between the accident and the beginning of treatment. If the case is seen immediately following prolapse and before any great amount of swelling has developed, and in those cases where there is no tearing of muscles or rupture of the optic nerve,

replacement into the orbit is usually possible and easily accomplished by means of moderate, even pressure over the eye-ball.

Occasionally it is necessary to enlarge the opening in the lids with an incision in the external canthus. Following the replacement, the eye should be immediately bandaged with the lids closed and maintained in this position for at least twenty-four hours. Cold packs under the bandage are beneficial. Where extreme swelling has occurred, or in those cases in which the muscles have been torn beyond repair, or the optic nerve severed, complete removal of the eye-ball, of course, is the only possible treatment.

### Enucleation

Enucleation should be done in such a manner that not only the eye-ball itself but the entire conjunctival sac, tear-gland and a portion of the tear-duct should be removed. The technic of this, briefly, consists, first in an incision completely surrounding the margin of the lids and parallel to them, approximately one-eighth inch from the edge. This incision is made through the skin only, and discission of the conjunctival sac from the skin follows. This is carried all the way around and down to the base of the orbit. Rather profuse hemorrhage occurs, chiefly in the vicinity of the inner canthus, and it is often well to anticipate this and check at least a portion of this by injecting adrenalin in this vicinity, prior to the beginning of the operation. Following the complete lifting-out of the eye and the conjunctiva, together with the entire edges of both lids, the cavity is filled with a sterile gauze pack and the edges of the wound closed with continuous suture, space being left at the inner canthus for the romoval of the pack the following day.

After-treatment consists of removal of any existing blood-clots and maintaining of antiseptics within the cavity. For this purpose, I prefer acriflavine jelly, which can readily be injected through the drainage opening. It is not irritating, highly antiseptic and promotes rapid granulation. Sutures can usually be removed about the fifth day. There are many conditions other than prolapse which call for enucleation and in them the technic is much the same as just described, except that in those cases where it is possible to close the lids over the eye prior to operation, this can be done, the edges of the lids being sutured together prior to the first incision and the sutured edges then grasped with forceps. This merely makes easier the discission of the conjunctiva from the skin.

# ANESTHETICS FOR SMALL ANIMALS: THEIR INDICATIONS AND USES*

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Surgery in small animals approaches more closely to human surgery than any other in the sphere of the veterinary surgeon, and the use of anesthetics for these animals has attained a high level of excellence. Anesthetics should be administered prior to the commencement of all operations involving severe or protracted pain. Not only is their employment prompted by humane considerations, but without it the accurate conduct of delicate operations is rendered a matter of great difficulty and often an impossibility owing to struggling on the part of the animal.

In the choice of an anesthetic the safety of the patient must always be the first consideration, for it is valueless to do a classical operation if the patient dies from the anesthetic. The surgeon must always take into consideration the age and condition of his patient, the severity of the operation, the chance which the animal has had of being properly prepared, and other circumstances which may occur at the time, as to the type of anesthesia he will use. For example, in certain cases of accident or other cause of urgency, the patient may be weak and faint from loss of blood and the use of general anesthetics is inadvisable. Their use is contraindicated also when cardiac or pulmonary diseases exist.

## GENERAL ANESTHETICS

From the general anesthetics we may select chloroform, ether, A. C. E. mixture, ethyl chloride, nitrous oxide gas, chloral hydrate, nembutal '844', or the narcotic, morphin sulfate.

Ethyl chloride and nitrous oxide gas are not used to any great extent in veterinary practice, partly because of the expense of the drug and the apparatus required for administration, but also due to the ease and convenience with which other anesthetics are used. Chloroform and ether, either alone or combined and diluted with ethyl alcohol, are the drugs most extensively used for the production of general anesthesia in small animals.

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The vapor of chloroform, if administered properly, is easily one of the best general anesthetics we can use. Its principal advantage is production of profound narcosis, preceded by a mild preliminary period of excitement, succeeded by rapid recovery from its effects. The chief factor in the causation of sudden death from chloroform is stimulation of the vagus which is the depressor nerve of the heart. If atropin sulfate (dose varying from 1/100 to 1/20th grain) is given 30 minutes to one hour before chloroform is used, the vagus nerve is paralyzed, which prevents its stimulation by chloroform, so that cardiac arrest does not follow. Another important point to remember with chloroform is to use it from a bottle that is freshly opened, as there is danger in using chloroform from a bottle that has been opened a number of times. Chloroform exposed to air decomposes into a very poisonous fosgin and chlorin gas.

If a therapeutic dose of morphin sulfate is given subcutaneously at least one hour subsequent to the administration of chloroform, the anesthesia is safer, more complete and we do not have the fear, struggling and shock of the first stage of inhalation anesthesia. Morphin sulfate is the chief alkaloid of opium and in the dog is one of the most perfect narcotics available. The dog has an idiosyncrasy to morphin which permits it to become readily narcotized and there is still a wide range of safety in its use. For many of the operations upon dogs, it gives the desired degree of anesthesia but in some cases a small dose of chloroform or ether is necessary or it may be combined with some local anesthetic.

Lacroix states that the dog under one year and over six months of age will require one-half the amount of morphin sulfate that is necessary to narcotize an animal that is of middle age or aged. Thus, give a 20-pound Collie puppy, three months of age, one-half grain of morphin sulfate, expecting that pup to be limp in an hour. The 25-pound Airedale, nine months of age, will require about one grain of morphin sulfate and the ten-year-old Fox Terrier, weighing 20 pounds, will require two grains.

Ether is safer than chloroform on account of its stimulating properties, but the stage of excitement is great and prolonged. It is also apt to cause undesirable after-effects in the form of affections of the respiratory tract. Under ether anesthesia there is always a great secretion of saliva and mucus, while under chloroform the amount is slight. Hence, in the administration of ether the position of the head is a matter of great importance and must

be such as to permit of drainage of the buccal secretions. For the safe administration of either of these drugs, a certain proportion of air is necessary. This is particularly true of chloroform, to which a large admixture is essential for safety.

On account of the depressant action of chloroform and the excitant action of ether, it was believed that the narcosis could be increased and the effect on the circulation better controlled if the two drugs were mixed. Various proportions have been tried, the English or A. C. E. mixture, consisting of ethyl alcohol one part, chloroform two parts, and ether three parts, is an excellent one. In the first 60 to 75 seconds, all the ether, with some chloroform, is evaporated; in the next three or four minutes, chloroform and alcohol, and in the following two minutes, the rest of the alcohol.

In anesthetizing cats, it is necessary to omit morphin sulfate, as in these animals it has a stimulating rather than a depressing effect. However, cats succumb to the effects of ether so easily, quickly and safely that there is little object in trying chloroform or any mixture of it.

Chloral hydrate is one of the most powerful central nerve depressants that we have and is very valuable to control the convulsions of strychnin poisoning, as the action is of long duration. One dram of chloral hydrate crystals dissolved in two ounces of water and administered per rectum is usually sufficient for a 20-pound dog having convulsions due to strychnin.

For general anesthesia or a hypnotic effect the one dram of chloral hydrate should be dissolved in four ounces of water and the 20-pound dog receive enough of this per rectum to produce the desired effect. Chloral hydrate has never been very widely used as a general anesthetic, due to the method of administration and uncertainty of its action.

Nembutal '844,' a new proprietary anesthetic preparation, seems to be well adapted for use in small animals. The dosage must be prepared accurately and is based on the live weight of the animal, using one-fifth grain per pound, making up a 5 per cent solution. It may be administered intravenously, when the action is rapid, beginning in approximately 5 minutes. Intraperitoneally or intramuscularly, its action will occur in 15 to 25 minutes. If given orally or per rectum, a littie longer time is required. In dogs an adequate surgical anesthesia with complete relaxation occurs for 2 to 2½ hours, with recovery in about 4 hours. In cats the action of the drug may persist for 8 hours or even longer.

Animals usually succumb to the effects of this drug easily and quietly. There is a decided decrease in the respirations, and they recover without any undesirable after-effects.

### Local Anesthetics

From the drugs which act as local anesthetics, we now have an excellent choice. The chief point to consider is the relative toxicity of the drug. The local anesthetic effect of some of these drugs is wonderful, their effect depending upon, first, the anesthetic being deposited in the proper place and, second, sufficient time being allowed to elapse befor the operation is commenced.

Local anesthetics may be used in the performance of major operations where the use of a general anesthetic is contraindicated. Such operations as laparatomy, cesarean section, ovariectomy, dental operations, reduction of hernias, caudectomy, amputations, extirpation of tumors, and castration may be performed.

Of the local anesthetics procain is probably one of the best. Its chief advantages are that it is comparatively non-toxic, non-irritating, and reliable in action. The local anesthetic action may be prolonged by the addition of adrenalin chloride (1-1000), using one cubic centimeter to each 100 cubic centimeters of the anesthetic solution.

Previous to using a local anesthetic for a long-continued operation or in very nervous small animals, it is well to use a preliminary dose of morphin sulfate or chloral hydrate.

There are many excellent local anesthetic proprietary preparations on the market.

To produce surface anesthesia of mucous membranes, cocain and butyn are the most efficient. Butyn in a two per cent solution seems to be best adapted for eye work, as if is less drying and, therefore, less irritating.

Epidural anesthesia may be used in operations on the posterior portion of the body and consists in blocking the nerve-trunks within the spine but outside the dura mater.

In introducing a local anesthetic into the epidural space, one of the most important points to remember is to inject it slowly and without pressure. Procain dissolved in a physiological salt solution to the extent of 2 per cent has been the preferred anesthetic because the toxic action is less than from any other known anesthetic substance and it is reliable in action. For dogs,

one cubic centimeter of a 2 per cent procain solution for each five pounds of body weight gives the desired amount of anesthesia.

Surgical anesthesia ordinarily takes place in 5 to 10 minutes and lasts approximately 45 minutes to one hour.

A glass syringe is preferred to any other, as it transmits more delicately the amount of pressure which is being used to make the injection which is of utmost importance to the operator in determining if the needle is properly placed. The injection of the solution should require no more pressure than if it was being injected into a free space.

The thinner the needle the less the trauma to the tissues, but the gauge of the needle should be compatible with its length. For dogs, a 20-gauge needle, two inches long, is preferred. In the dog, epidural anesthesia is accomplished by inserting the needle between the last lumbar vertebra and the sacrum. The point to insert the needle may be located by palpating for the depression just anterior to the first sacral vertebra, or the depression may be located on a line drawn transversely across from the posterior borders of the wings of the ilium. It is preferred that the animal remain in the standing position so that the anatomical structures will be in normal position.

The needle should be inserted exactly in the center of the depression and tilted slightly backward. The depth the needle will have to be inserted will vary from one-half inch in small dogs to two inches in large dogs, weighing over 100 pounds.

If it is desired to operate in the abdominal cavity, the posterior parts should be slightly elevated, so that the anesthetic solution will come in contact with the nerves supplying that region.

This method of anesthesia has been used for cesarean section, spaying, castration, amputation of portions of the hind legs, and reduction of fractures. For caudectomy the amount of anesthetic solution necessary will vary from one-half cubic centimeter in young puppies to two cubic centimeters of the 2 per cent procain solution in older dogs.

Epidural anesthesia in the cat is accomplished in the same manner as in the dog. The depth the needle will have to be inserted will vary from ½ to ¾ of an inch. Mature cats will require three cubic centimeters of the 2 per cent procain solution, while the dose for younger cats will be smaller, depending upon the size.

Nerve-blocking of the infraorbital and mandibular alveolar nerves should be a common procedure for painful operations on

the teeth of dogs. The dental canals are readily accessible, so it is not a difficult process.

The infraorbital nerve is a branch of the maxillary nerve and contributes dental branches to the upper teeth. After emerging from the infraorbital foramen, it supplies the upper lip and nose with filaments. The teeth and structures in the lower jaw are supplied by branches from the mandibular alveolar nerve.

A 2 per cent solution of procain is preferred, because it is reliable in action and comparatively non-toxic. The instruments needed are a glass syringe and a 20-gauge needle two inches long. The skin at the point of insertion of the needle should be disinfected and an insensitive wheal produced.

The infraorbital nerve may be blocked at two points. One method is to select a point about 1½ inches below the lateral canthus of the eye, in the space between the posterior border of the malar bone and the anterior border of the coronoid process of the mandible. The needle is inserted vertically through the skin and pushed through the soft tissues in the space between the anterior border of the coronoid process of the mandible and the malar bone until its point has passed the edge of the latter. It is then directed forward along an imaginary line that would reach the gingival margin of the upper incisor teeth until the point of the needle reaches the maxillary foramen where the nerve is lodged and the injection is to be made. This is at a depth of approximately 1 to 1½ inches from the surface in a dog of average size.

Another method, that of Jean-Fernand, is to locate the infraorbital foramen, which is easily accomplished in the dog, and to insert the needle along the floor of the infraorbital canal for a short distance. At either point inject two cubic centimeters of a 2 per cent solution of procain. Anesthesia will be complete in 10 minutes and last for approximately 20 minutes.

The mandibular alveolar nerve may be reached very easily to be injected. Pass the finger along the lower border of the mandible, from front to back, to a distinct depression. The point at which to insert the needle is at the lowest part of the depression. Insert the needle directly upward, close to the medial surface of the mandible, for a distance of ½ to ¾ of an inch and inject two cubic centimeters of a 2 per cent procain solution. Anesthesia will take place as described above.

# STUDIES OF TUBERCULOSIS OF TURKEYS*

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During July, 1930, one of the writers (W. H. B.) noted considerable discrepancy between the results of the tuberculin test and the autopsy findings in a flock of turkeys which he was called upon to test in Lassen County, California. These observations were reported to both the California Agricultural Experiment Station and the Nevada Veterinary Control Service, because the ranch on which the outbreak occurred was near the California-Nevada state line.

Recognizing a need for information regarding this disease as it affects turkeys and because the problem was one which is of equal importance to turkey-growers of both California and Nevada, a coöperative field investigation was made of the Lassen County outbreak. Arrangements were made with the owner of the flock to use it for experiment purposes, and a coöperative project on testing was outlined. Later, the investigations were extended to include other phases of the disease, and the results reported in this paper are in the nature of a progress report of the studies made to date. They are presented for the purpose of record and for discussion.

## EPIZOOLOGY

*Distribution in California and Nevada:* The diagnostic records of the various laboratories in these states were examined to determine the extent and distribution of tuberculosis among turkeys in the two states. These records indicated that the disease is not of great economic importance and apparently exists at present only among turkeys reared in sections where the disease exists also among chickens. The facts that the disease was found to exist in both states, that there is an increasing tendency toward using older turkeys for breeding stock as turkey husbandry methods improve, and that the traffic in adult breeding stock is increasing, make the problem an important one for consideration.

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†Resigned, May 31, 1931.

Visits made to five ranches where the disease had been diagnosed among turkeys revealed conditions which indicated that tuberculosis among turkeys is associated with the confined or barnyard flocks rather than with the range flocks. Chickens were found on four of the five ranches; on the fifth the breeding turkeys in which tuberculosis was diagnosed had been transferred recently from a ranch where they were being ranged with chickens known to have had tuberculosis.

*Age incidence:* It is now generally recognized that the incidence of tuberculosis in chickens increases with the age of the flock. Field observations of the disease among turkeys indicated that this fact was equally true in turkey flocks. Careful autopsies and bacteriological examinations of 88 turkeys from four infected ranches were made to determine the presence or absence of lesions and of acid-fast bacilli. A summary of these results, classified according to the age of the turkeys, appears in table I.

Although these data are limited, they suggest that there is a greater prevalence of tuberculosis among the older turkeys. In the group recorded as less than one year old, the majority of the birds were from 6 to 10 months old. The one infected bird in this group was 10 months old. In the unknown-age group, the majority probably belong in the one-two-year group, but the exact age was not known.

In Nevada, where chicken hens were reported by Niemann[1] to be commonly used by turkey-producers for hatching and brooding poults, the incidence of tuberculosis among chickens was found to be greater than among turkeys, in spite of the intimate association. The fact that the majority of the market poults are slaughtered before they reach one year of age and that most of the turkey hens used for breeding purposes are sold for slaughter before they reach 18 months of age probably accounts for this lower incidence among turkeys, while there is a tendency to keep chickens until they are several years old. In California there is more of a tendency to keep older turkeys, but the avian-tuberculosis areas are not, as a rule, turkey-growing areas, which fact undoubtedly helps to reduce the incidence of the disease among turkeys in this state.

*Types affecting turkeys:* Typing tests on tuberculous material from five turkeys secured from three different sources in California have been completed. In each of these cases, two rabbits, two guinea pigs and two chickens were inoculated intra-peritoneally with one cubic centimeter of a saline emulsion prepared

from tuberculous organs. Unless death intervened, all the animals were kept for a period of at least three months before being killed for autopsy. The chickens came from a flock which has been under constant supervision and tuberculosis has never been diagnosed in chickens from this ranch. Chickens under one year of age were used whenever available and when older birds had to be substituted, they were first subjected to the tuberculin test. The rabbits and guinea pigs also were tested previous to inoculation in three trials.

Four of the cases from three ranches were found to be unquestionably of the avian type of tuberculosis, according to the classification used by Van Es and Martin.[2] A saline emulsion of a tuberculous liver from the fifth turkey, which originated from one of the same ranches, did not react so typically in the laboratory animals, although reinoculation indicated that it was probably suffering also from the avian type. A summary of the results of inoculation of laboratory animals with material from the fifth turkey follows:

*Chickens:* One was killed at the end of 90 days and was found to be normal on autopsy. The second died of tuberculosis on the 81st day. Inoculation of a second series of laboratory animals with material from this chicken resulted in the development of tuberculosis in the two chickens, neither of which died in 90 days. Both guinea pigs in this second series remained normal, and the rabbits died prematurely of coccidiosis.

*Guinea pigs:* One died in 20 days with lesions in the liver, spleen and at the point of inoculation. Acid-fast organisms were demonstrated. The second died in 25 days but lesions and acid-fast rods were found only at the point of inoculation. Two guinea pigs inoculated with a saline emulsion of the lesions from the second pig died prematurely from a paratyphoid infection which was accidentally introduced into the colony soon after the inoculations were made.

*Rabbits:* Both rabbits were alive after four months. One was normal on autopsy and the other was found to be suffering from coccidiosis of the liver, with questionable lesions of tuberculosis. No acid-fast rods were demonstrated in smears made from these rabbits. However, two chickens and two rabbits were inoculated with emulsions of the liver of the second rabbit. One of the chickens died prematurely from trauma of the gizzard due to a wire piercing it, but the other chicken had generalized tuberculosis when killed at the end of 83 days. The rabbits were killed after 90 days and were found to be suffering from mild cases of tuberculosis of the livers and spleens. Acid-fast rods were demonstrated.

Although the tuberculous material from one turkey did not type true to the avian form in the original tests, the results obtained in these few trials would indicate that tuberculosis in turkeys is usually of avian origin. Van Es and Martin[2] have typed two cases of tuberculosis in turkeys and found both to be of avian origin. As far as is known to the writers, there are no cases on record of turkeys suffering from either the human or the bovine type of tuberculosis.

TABLE I—*Age incidence of tuberculosis in turkeys from infected flocks.*

| AGE | TOTAL NUMBER OF TURKEYS | TUBERCULOUS | | NON-TUBERCULOUS | |
|---|---|---|---|---|---|
| | | NUMBER | PER CENT | NUMBER | PER CENT |
| Unknown..... | 10 | 2 | 20.0 | 8 | 80.0 |
| Less than one year | 11 | 1 | 9.09 | 10 | 90.91 |
| 1 to 2 years..... | 24 | 14 | 58.33 | 10 | 41.66 |
| Over 2 years..... | 43 | 28 | 65.12 | 15 | 34.88 |
| Totals..... | 88 | 45 | 51.14 | 43 | 48.86 |

## SYMPTOMS

From the observations made thus far, the symptoms seen in the field are so meager that by casual inspection one would seldom suspect a flock of turkeys to be suffering from the disease. Lameness and emaciation have not been observed to be as common symptoms as in case of the disease among chickens. Tuberculous turkeys which were placed in individual cages and observed for periods of one to ten weeks usually held their initial weight and a few gained in weight. Two turkeys that died while under such observation showed symptoms that were typical of the advanced stages of tuberculosis in animals. In these two birds there were intermittent periods of normality and depression lasting from two to three weeks previous to death. In the periods of depression, which lasted for two or three days, the feathers became ruffled, the appetite diminished, and a diarrhea developed. These periods were followed by a few days of normal appetite and a general improvement of health.

Temperatures of two non-tuberculous and five tuberculous turkeys were taken over a six-day period at two-hour intervals from 8 a. m. to 8 p. m. Intradermal avian tuberculin, in approximately 0.05 cc doses, was injected in the wing-web of each turkey the second day. The individual temperatures were found to be exceptionally uniform except for variations which were statistically proven to be associated with the time of day. No change was apparent following the injection of the tuberculin nor was there a significant difference in the temperatures of the normal and infected birds.

## GROSS PATHOLOGY

The gross pathology of tuberculosis in turkeys has not been found to be markedly different from the disease as it affects chickens. Most of the autopsies which have been made by the

writers were on specimens killed for market purposes rather than ones that have died from the disease. This has afforded an opportunity to see turkeys in all stages of the disease and in most cases the external appearance of the bird has proved to be no indication of the extent or nature of the lesions.

*Distribution of lesions:* The lesions were found to be typical of those seen in chickens and their distribution has been similar.

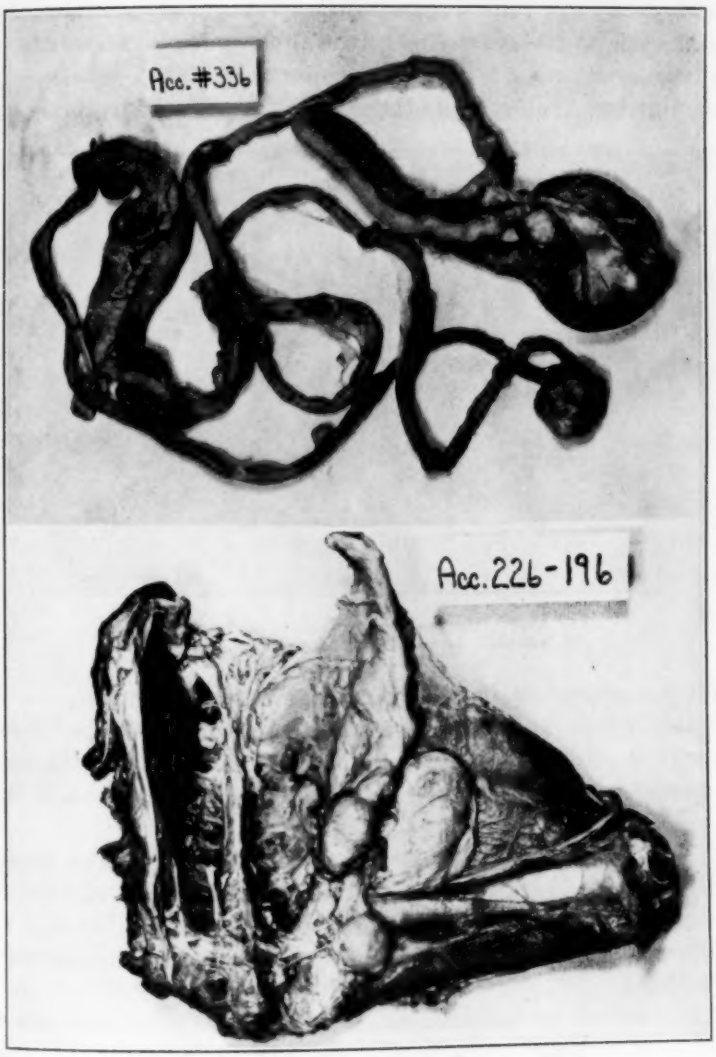


FIG. 1 (above). Lesions of tuberculosis in the intestine (turkey).
FIG. 2 (below). Lesions of tuberculosis in the muscles (turkey).

A summary of the distribution in 46 tuberculous turkeys is given in table II. For comparison the distributions in chickens reported by Bushnell and Hinshaw[3] and by Vawter[4] also are included in this table. Those reported by Bushnell and Hinshaw were from Kansas outbreaks and those of Vawter from Nevada outbreaks.

The ovaries in the turkeys examined were not an uncommon seat of infection and the lesions seen in the ovaries were similar to those seen in other organs, although the lesions were seldom larger than a hickory nut. In the functioning ovaries only the smaller undeveloped ova were abnormal. The lesion in the one infected oviduct was attached to the serous membrane and

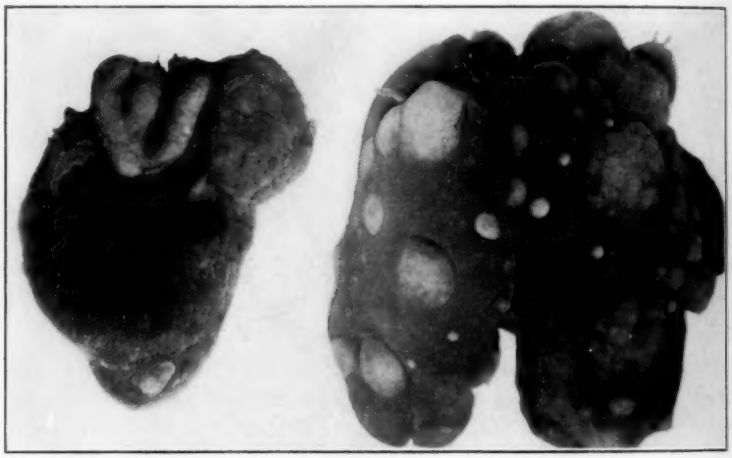

FIG. 3 (left). Lesions of tuberculosis in the heart (turkey).
FIG. 4 (right). Lesion of tuberculosis in the liver (turkey).

did not appear to have any opening into the lumen. In the turkey which had infected testes there were no other lesions except a marked atrophy of the testes. Smears from the testes contained large numbers of acid-fast bacilli, while smears from the liver, spleen and lungs were negative.

Nine turkeys of 31 examined showed lesions in the thymus gland. These varied from the size of a pea to that of a marble and numbered from one to three to a specimen. It was not difficult to palpate these lesions by an external examination of the gland.

The lesions in the muscles could probably more correctly be classified as being in the fascia. In each case the muscle lesions were in the posterior abdominal region.

The one skin lesion was attached to the subcutaneous tissue in the region of the crop. Only a few crops were examined and are not reported in the table, but one case was observed in which there was a tuberculous ulcer of the mucous membrane of the crop. This ulcer was teeming with acid-fast bacilli.

It would appear from the distribution of lesions in the turkeys examined that there is a tendency for a greater number of organs to become infected than in chickens. It is evident, however, that, as in chickens, the disease is principally abdominal in nature.

TABLE II—*Comparison of distribution of lesions of tuberculosis in turkeys and chickens.*

| LOCATION OF LESIONS | CHICKENS | | TURKEYS | | |
|---|---|---|---|---|---|
| | VAWTER* (PER CENT) | BUSHNELL† (PER CENT) | SPECIMENS | LESIONS | |
| | | | | NUMBER | PER CENT |
| Liver | 87.7 | 97.0 | 46 | 44 | 95.65 |
| Spleen | 61.2 | 97.0 | 46 | 31 | 67.39 |
| Intestine | 59.1 | 75.0 | 46 | 21 | 45.65 |
| Lungs | 49.0 | 27.0 | 46 | 15 | 32.61 |
| Thymus | .... | .... | 31 | 9 | 29.03 |
| Mesentery | .... | .... | 46 | 10 | 21.74 |
| Ovaries | .... | 0.0 | 31 | 10 | 32.26 |
| Testes | .... | .... | 4 | 1 | 25.00 |
| Pancreas | .... | .... | 31 | 4 | 12.90 |
| Muscle | .... | .... | 17 | 2 | 11.76 |
| Bones | 2.+ | 18.0 | 27 | 2 | 7.41 |
| Skin | .... | 6.0 | 16 | 1 | 6.25 |
| Gizzard | 2.+ | 1.0 | 35 | 2 | 5.71 |
| Esophagus | .... | .... | 21 | 1 | 4.76 |
| Proventriculus | .... | .... | 27 | 1 | 3.70 |
| Kidneys | .... | 15.0 | 28 | 1 | 3.57 |
| Oviduct | .... | .... | 33 | 1 | 3.03 |
| Pericardium | .... | 0.0 | 44 | 2 | 4.55 |
| Myocardium | .... | 0.0 | 44 | 1 | 2.27 |
| Epicardium | .... | .... | 44 | 0 | 0.0 |
| No visible lesions. | 4.0 | .... | 46 | 0 | 0.0 |

*49 birds.
†100 birds.

## DIAGNOSIS

The discovery of tuberculosis in turkey flocks, in the experience of the writers has, been made more often by autopsy by the owner of a bird that has died of the disease, and by the discovery by a housewife of a tuberculous specimen when preparing a bird for table use, than by symptoms seen in the flock or by the use of the tuberculin test. In the Lassen County flock referred to in the introduction, the disease was discovered by means of the tuberculin test after it was learned that the turkeys

had been associating with chickens that were known to be tuberculous.

*The tuberculin test:* It is principally with the use of the tuberculin test that this discussion of diagnosis is concerned. At the time the Lassen County flock was tested the efficiency of the tuberculin test was questioned when non-reactors were found to be tuberculous on autopsy. The original test on this flock was made in July, 1930, with a commercial avian intradermal tuberculin, and the injections were made in the wattle, the loose pendulous fold of skin on the neck, sometimes spoken of as the dewlap. There were five reactors in the flock of 105 turkeys and between 40 and 50 reactors in the chickens tested with the same lot of tuberculin. In the majority of the tests reported in this paper, a 26-gauge needle was used for making injections. In the other instances a 27-gauge needle was substituted.

September 15, the writers retested this flock of turkeys, using a commercial avian intradermal tuberculin which had been tested previously on known tuberculous chickens. Injections were made in the wattle in all of the 58 turkeys which remained in the flock. In 48 of these birds, injections were made also in the skin of the edge of the wing-web. This procedure necessitated pulling a few feathers before injecting the tuberculin.

The injection in the wattle was considered satisfactory when a pale bleb of three to five millimeters in diameter appeared. In the wing the tuberculin appeared to pass readily between two layers of the skin and a transparent bleb formed. Readings were made 48 hours after injecting and all reactors which were not immediately killed for autopsy were segregated and the reactions checked at the end of 72 hours. All of the reactors were leg-banded and the reactions recorded, and all the non-reactors were identified with a tattoo mark.

There was no agreement in the reactions in the two areas of injection. Only one turkey gave a typical wattle reaction, while 23 gave typical wing-web reactions. Arrangements were made for purchasing part of the reactors. Part of the reactors purchased were killed immediately and examined on the ranch, a few were taken to Reno, and the rest were shipped to Davis. The turkeys which were shipped to Davis were immediately placed in individual cages and held for observation and further testing before autopsy.

The remainder of the flock was sold through the courtesy of the California Turkey Growers' Association, with the privilege

of inspection at the time of killing and dressing. These turkeys were killed and picked at the ranch, November 8, and shipped to San Francisco as soon as they were chilled enough to pack. November 10, one of the writers (W. R. H.) went to San Francisco and inspected each bird as it was drawn and made notes of all lesions observed. Each examination consisted of an inspection of all the viscera, a palpation of the carcass for skin and thymus lesions, and an inspection of the body cavity with the aid of an electric light for bone, joint and other lesions within the cavity. All suspicious lesions were placed in specimen boxes, labeled with the number of the bird for identification, and taken to the laboratory at Davis for microscopic examination for acid-fast bacilli. At least two smears were made from each specimen and unless acid-fast organisms were readily found, at least 50 fields on each smear were examined before the bird was considered non-tuberculous.

TABLE III—*Comparison of autopsy findings with the results of tuberculin tests in turkeys*

| Autopsy Findings | Wattle Reactions | | | | | Wing-Web Reactions | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Positive | | Negative | | Total Number of Turkeys | Positive | | Negative | | Total Number of Turkeys |
| | Number | Per Cent | Number | Per Cent | | Number | Per Cent | Number | Per Cent | |
| Non-tuberculous.. | 1 | 2.77 | 35 | 97.22 | 36 | 13 | 30.95 | 29 | 69.05 | 42 |
| Tuberculosis.... | 5 | 11.11 | 40 | 88.88 | 45 | 28 | 75.68 | 9 | 24.32 | 37 |

The results are given in table III. It was evident from the autopsy results that the wattle of the turkey is not a desirable site for injecting tuberculin but that the wing-web holds possibilities of being a suitable site of injection. To obtain further data, arrangements were made with three other flock-owners to make similar tests. The number of birds which were available for autopsy in these flocks was small but the combined information from the four flocks furnished the data for the summary given in table IV.

In all, 88 turkeys from the four flocks were examined for lesions, 81 of which had been tested by the wattle method and 79 by the wing-web method. The reactions in the wattle agreed with the autopsy results in 97.22 per cent of the non-tuberculous birds but in only 11.11 per cent of the tuberculous birds. Considering the

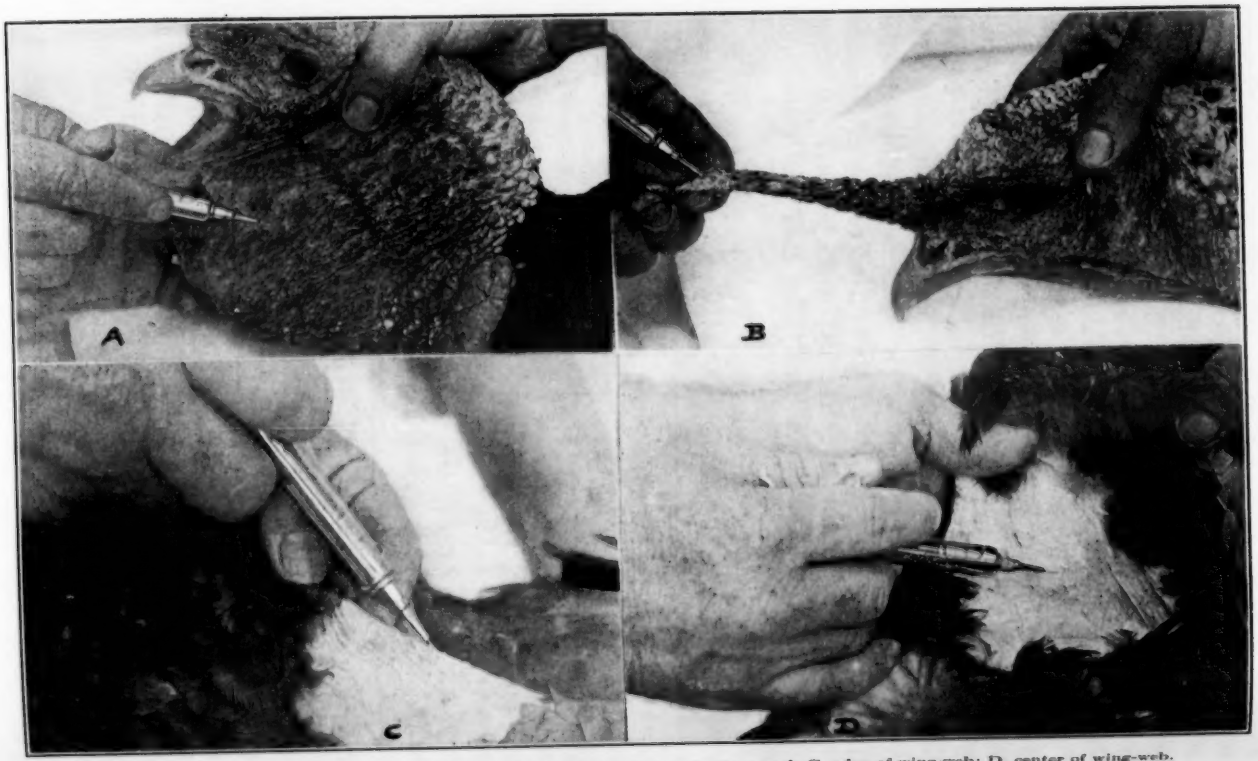


FIG. 5. Possible areas for injection of tuberculin in turkeys. A, wattle; B, snood; C, edge of wing-web; D, center of wing-web.

total agreements, the efficiency of the wattle as a site for injection was found to be 49.38 per cent.

Similar comparisons made with the wing-web injections showed that the reactions in the non-tuberculous birds agreed in 69.05 per cent of the cases, while in the tuberculous birds there was an agreement of the reactions and autopsy findings in 75.68 per cent of the birds. The total agreement with autopsy findings was 72.15 per cent. It is evident, therefore, that in these cases the wing proved to be the more efficient site of injection but it also falls short of perfection.

TABLE IV—*Summary of autopsy findings compared with results of tests.*

| AUTOPSY FINDINGS AND TUBERCULIN TESTS | WATTLE REACTIONS | | WING-WEB REACTIONS | |
|---|---|---|---|---|
| | NUMBER | PER CENT | NUMBER | PER CENT |
| Agreed | 40 | 49.38 | 57 | 72.15 |
| Disagreed | 41 | 50.62 | 22 | 27.85 |
| Total turkeys | 81 | 100.00 | 79 | 100.00 |

A histological study of injections of tattoo and India ink made in the same manner as tuberculin injections showed in agreement with Bushnell and Brandly,[5] that little if any of the material was injected in the skin of the wattle. In the case of the wing-web, opportunity for depositing the material intradermally seemed considerably greater due to the "natural" separation of what appeared to be the pars papillaris and the pars reticularis of the derma. This was evident when inoculations were being made in the skin of the wing-web, and accounts for what has been called, earlier in this paper, "a transparent bleb" in the inoculation of tuberculin. In other words, when the needle is inserted and the tuberculin is forced out of the syringe into the skin, a bleb rises and the needle-point becomes visible in the tuberculin.

Other points of injection that have been suggested are the snood (the loose process on the anterior dorsal portion of the head) and the mucous membrane of the anus. In a limited number of trials, neither of these areas has offered much as a possible site of injection.

*Differential diagnosis:* Some of the conditions noted in turkeys which might have been confused with tuberculosis are mycosis, blackhead and tumors. Mycotic lesions in the liver and kidney, which on first glance were suggestive of tubercles, have been observed. These were not definitely capsulated and

circumscribed, however, and on microscopic examination, mycelia were found, while acid-fast rods were not demonstrated.

Blackhead or infectious entero-hepatitis should not be confused, as the lesions in the liver do not resemble tubercles. Furthermore, the well-known characteristic lesions of the disease in the ceca should help to differentiate it from tuberculosis. On the other hand, tumors of the liver and ovary have been noted that were suggestive of tuberculosis until microscopic examination failed to reveal acid-fast rods.

## Summary

Preliminary studies of tuberculosis in turkeys in California and Nevada are reported.

Tuberculosis in turkeys in these states was found to be of no economic importance except in areas where the disease is prevalent among chickens. The disease was observed only in closely confined or barnyard flocks of turkeys, and the incidence increased with the age of the turkeys.

Four cases of tuberculosis of turkeys from three outbreaks in California were found to be of avian origin by inoculation of tuberculous material into chickens, guinea pigs and rabbits. A fifth case originating from one of the three outbreaks did not produce typical avian reactions in the original inoculation of laboratory animals, but subesquent tests indicated that it was of avian or possibly mixed origin.

The classification of lesions in 46 tuberculous turkeys indicated that the disease is of abdominal origin. The liver, spleen, intestine, lungs, ovaries and the thymus gland were found, in the order given, to be the most common seats of infection. One case was studied in which the testes were the only seat of infection. By a comparison of lesions in the turkeys examined, it would appear that there is a tendency for a greater variety of organs to become infected than in chickens. In the cases observed, the external appearance or condition of the turkey was no indication of the extent or nature of the lesions found on autopsy.

Based on autopsy findings in 88 turkeys from four ranches, the intradermal tuberculin test was found to be a less desirable means of diagnosing tuberculosis in turkeys than in chickens. The accuracy of the test in turkeys was greater when injections were made in the skin of the wing-web than when made in the skin of the wattle. The wattle reactions compared with the autopsy findings in 49.38 per cent of the cases in contrast to an

agreement of 72.15 per cent in wing-web injections. In the tuberculous turkeys the difference was greater, as the wattle reactions agreed with the autopsy findings in only 11.11 per cent of the cases, as compared to an agreement of 75.66 per cent of the wing-web reactions and autopsy findings.

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# A MODIFICATION OF THE RAPID AGGLUTINATION TEST FOR PULLORUM DISEASE*

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In 1929, Bunyea, Hall and Dorset[1] reported on the use of a rapid agglutination test for pullorum disease designed for use with fresh whole blood. They suggested also that a drop of blood, taken up on filter paper and dried, could be tested later in the laboratory, by the same method. We used both methods, but with indifferent results, being bothered by poor light and working facilities in the poultry-house and by mechanical difficulties in the filter-paper method.

In December, 1930, at the Conference of Research Workers in Animal Diseases, at Chicago, Dr. Bunyea reported on the testing of whole blood with a stained antigen, a rather heavy antigen stained deeply with methyl or crystal violet. Dr. M. Dorset sent us a supply of this stained antigen to try out in routine poultry testing, checking results against the serum-tube method. We used some of this antigen on blood dried on filter paper, but again had poor results, due to faulty technic. Early in February, we tried blood-smears on ordinary microscopic slides, with much better results.

On five small flocks, we used the three tests: the serum-tube test, the fresh whole-blood, and the dried blood-smear test, using the stained antigen in the last two.

TABLE I—*Comparison of three tests.*

| FLOCK | BIRDS | REACTORS | | |
|---|---|---|---|---|
| | | TUBE | WHOLE BLOOD | DRY SMEAR |
| 1 | 171 | 31 | 29 ( 93%) | 30 (97%) |
| 2 | 66 | 11 | 9 ( 82%) | 9 (82%) |
| 3 | 318 | 56 | 58 (100%) | 54 (96%) |
| 4 | 82 | 29 | 29 (100%) | 26 (89%) |
| 5 | 117 | 12 | 11 ( 91%) | 10 (84%) |

Assuming the serum-tube method to be 100 per cent, the dried blood-smear test in these five flocks gave an average accuracy of 90 per cent. This average is lower than it should be on account

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of the small number of reactors in flocks 2 and 5, where a discrepancy in two reactors cut down the average by 18 and 16 per cent respectively.

On six flocks we compared the results of the wet and dry blood-smears. Compared with the fresh-blood test, the dried blood-smear, even in our inexperienced hands, averaged 94 per cent in accuracy.

TABLE II—*Comparison of fresh-blood test with dried blood-smear.*

| FLOCK | BIRDS | REACTORS | |
|---|---|---|---|
| | | FRESH BLOOD | DRY BLOOD |
| 1 | 112 | 26 | 24 ( 92%) |
| 2 | 188 | 22 | 22 (100%) |
| 3 | 169 | 11 | 11 (100%) |
| 4 | 61 | 26 | 24 ( 92%) |
| 5 | 162 | 36 | 34 ( 94%) |
| 6 | 688 | 176 | 167 ( 95%) |

Since completing the above tests, our attention has been called to an article published by Green and Robinson,[2] who report good results with dried blood-smears, although they used the unstained antigen.

As yet, we have made no attempt to determine the dilution, or the serum-antigen relation in the dry smear. The blood-smears vary a great deal in thickness and area covered, and although we use a standard drop of antigen, the amount of blood used is difficult to determine. Without doubt, this wide variation in the dilution is partly responsible for the discrepancy of nearly 10 per cent between the dry-smear and the tube test. Dr. Dorset suggested the use of a standard wire loop for transferring blood to the slide, to establish a definite dilution, but we have had no opportunity to try it.

Our technic consists of making a needle puncture in the ulnar vein, transferring a small drop of blood to a labeled microscopic slide, and making a blood-smear slightly thicker than the smear ordinarily made for blood study. If the smear is too thick, or the blood allowed to coagulate as a drop, and not smeared at all, it will flake off on drying, and be lost. A properly made smear will not flake off at all. We write the leg-band number on the label of the slide and pack in slide-boxes of one hundred slides each. These smears can be tested whenever it is convenient. We get exactly comparable results on duplicate slides; that is, making duplicate blood-smears from each hen, testing one set

the day after bleeding, and the others at periods from one to six weeks later.

In testing, we place eight or ten slides in a row on a glass plate elevated an inch or so above a white background. A drop of antigen is placed on each slide, picking out the most uniformly-spread portion of the blood-smear, and the blood and antigen mixed with the point of a dissecting needle, over an area the size of a penny. When the last slide has been handled, reactions will already have appeared in the first slides, and others may appear slowly as the slides are rocked from side to side. The reactors are laid to one side, and the non-reacting slides dropped into a jar of cleaning fluid.

It requires less than five minutes to test and read a string of ten or twelve slides, and it has been our experience that a longer wait for agglutination to take place has not made much difference. In many cases agglutination takes place almost immediately after the antigen is mixed with the serum. Using a deeply-stained antigen, a typical agglutination can hardly be overlooked. The dark purple masses of agglutinated organisms form large conspicuous flakes, the opaque solution becomes clear, and the floating fragments of dried blood do not confuse one. Where a partial agglutination is suspected, the slide can be viewed through the microscope to determine the nature of the suspected coagula.

This rather crude test may receive hearty condemnation by those who are working with flocks to be certified free from bacillary white diarrhea, and want 100 per cent results, and there are many factors of error. There is no opportunity to retest a blood sample; once a blood smear is tested, it is done with. Also, birds reacting in a very low dilution only, may be passed as healthy. Yet we believe that this test has a sphere of usefulness. It does away entirely with the no-serum, broken, hemolyzed, and otherwise unfit blood samples so often encountered in the serum test. There are no sample numbers so stained as to be illegible. Furthermore, the leg-band numbers are copied but once in the whole operation, doing away with considerable clerical error. This test does away with the endless washing, sterilization, and wear and tear on tubes and pipettes. Without sacrificing accuracy to speed, the laboratory worker can save two-thirds of the time consumed in making the serum-tube test. No apparatus is needed beyond a glass plate, a needle, and the antigen.

Last but not least, the dry-blood test eliminates the necessity of giving immediate attention to shipments of blood samples arriving at the laboratory, which have a way of arriving at the most inopportune times. A shipment of blood-tubes, reaching the laboratory at 5 p. m. Saturday, necessitates Sunday work. When such a shipment contains dry blood-smears, they can remain in their original package until Monday, or, if need be, until two weeks from Monday. In many states there is but one laboratory for this work and long distances to ship blood. In Montana, blood samples may have to travel five hundred or more miles, two or three days en route, and under very uncertain weather conditions. Shipping whole blood, except in specially-constructed containers, under such conditions, is nothing but a gamble.

Conceding that there is a possible 10 per cent error in using the dry-blood test, yet this method is sufficiently accurate for certain conditions. We have used it as an adjunct to culling flocks, both for selecting the breeding flock and for culling for increased egg-production. It can be used by poultrymen who do not especially desire a B. W. D.-free flock, but who would like to minimize their losses from B. W. D. The decreased cost of the test appeals to the flock-owner. We have roughly estimated that we can test by this method for 1½ cents per bird. We believe that with further use of the test, and by smoothing out some of the rough spots in the technic, that its accuracy can be greatly increased. In a small laboratory, with no especial provision made for testing large numbers of blood samples, in areas where most of the bleeding must be done by the owners, and especially in areas where long distances and uncertain weather conditions prevail, the use of this method is practicable.

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## Wins Scholarship

Malcolm S. Knowles, son of Dr. and Mrs. A. D. Knowles, of Palm Beach, Florida, and a sophomore at Harvard University, was recently announced as the winner of a scholarship to Geneva, Switzerland, this summer, to study foreign affairs in connection with the League of Nations, under the auspices of the Students International Union of New York.

# STUDIES OF SOME VIRUS DISEASES OF FOWLS*

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## INTRODUCTION

The so-called filtrable-virus diseases of fowls constitute a formidable economic hazard to the poultry industry of the world. Certain widely prevalent virus diseases, because of the filtrable character of the etiological agent and their frequent association with conditions and factors of obscure origin, admittedly are more difficult to control than diseases of known cultivable etiology. Members of the group of virus diseases of fowls (fowl-pox, tracheo-bronchitis, psittacosis, fowl-pest) typify many features of the virus diseases of man and other animals. Fowl-pox (contagious epithelioma, avian molluscum, avian or fowl diphtheria, canker, roup, infectious coryza, bird-pox), widely prevalent in Europe and America, has by reason of positive transmission tests of the virus to mammals by Zwick[1] and others, confirmed the conception that the poxes of the different domestic animals may be nothing more than local varieties of a generic pox, probably variola. Infectious tracheo-bronchitis (infectious bronchitis, infectious tracheitis, infectious laryngo-tracheitis) of fowls, recorded from the United States and Canada, resembles a number of the recognized virus entities of other species involving primarily or exclusively the respiratory system.

Recent and current investigations have fortified our knowledge of the two diseases mentioned and it is proposed herewith to consider briefly some of the important aspects and results of this work.

## OCCURRENCE AND TRANSMISSION

Fowl-pox, recognized as a separate disease entity by Bollinger,[2] in 1873, was shown by Marx and Sticker,[3] in 1902, to be caused by a Berkefeld-filtrable agent. Carnwarth,[4] in 1908, showed the causal identity of cutaneus pox and a form of avian diphtheria, while Uhlenhuth and Manteufel[5] reproduced comb and throat lesions by inoculation with throat and comb lesion material, respectively. In spite of evidence mitigating the importance of

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specific or non-specific avian diphtheria of bacterial origin, the mutual occurrence of various bacteria and pox virus in diphtheritic lesions may indicate an interrelationship yet to be clarified.

Infectious tracheo-bronchitis, although considered a relatively new disease of fowls, may have occurred sporadically for many years[6] before its recognition as a specific infection of epiornithic significance.[7] The belief that it was closely related to non-specific factors,[8,9] chiefly exposure to cold, dampness and improper feeding, has been largely disproved by positive transmission experiments with filtrates[10,13] (Berkefeld and Seitz) of tissue and respiratory exudates from typical cases. The influence of the elements and of lower temperatures on mortality among fowls affected with tracheo-bronchitis,[14] however, can not be overlooked.

Fowl-pox and tracheo-bronchitis affect primarily the domestic fowl. Both have been observed to occur less commonly in turkeys, pigeons and certain wild birds, including quail and sparrows. Tracheo-bronchitis has been reported as occurring in blackbirds and ducks and geese, while observations of natural cases of fowl-pox in these species have not been encountered in the literature. Inability to infect ducks with fowl-pox artificially has been reported by Doyle and Minnett,[15] while absence of the disease in this species in Holland, where pox among chickens is very common, was observed by teHennepe.[16]

The knowledge of the mechanism of pox dissemination has recently been fortified by the proof of insect transmission by means of mosquitoes.[17,18] Tracheo-bronchitis, essentially a disease of the respiratory tissues, is generally considered to be largely airborne. A greater susceptibility to spontaneous fowl-pox by the large-comb breeds of fowl has been observed frequently.[19,21]

A consideration of other epizoological factors related to the two diseases reveals an apparently equal susceptibility at all ages, unless previously exposed. A seasonal distribution or variation in the incidence of infection may largely, in the case of fowl-pox, be compatible with the periods of insect-vector activity and with acquired active immunity.[19] The seasonal trend of tracheo-bronchitis is toward late summer, fall and early winter, but in localities where chicks are primarily affected the seasonal incidence has been observed by Hinshaw[22] to be subject to change.

## MANIFESTATIONS AND COURSE

Fowl-pox is commonly manifested in two forms, a cutaneous (eruptive) and a diphtheritic (mucous membrane). Brumley and Snook[23] and Beaudette[19] observe that the skin type occurs with greater frequency during the warmer seasons of the year or in warmer climates, while the diphtheritic type predominates during the cooler months or in less temperate climates. A form characterized by muco-purulent exudative involvement of the oculo-nasal tissues is recognized by some investigators (Doyle,[24] Moussu[25]), presumably on the basis of infectivity of the discharges. Ordinarily, this coryza-like manifestation in cases of pox is considered secondary because of complication with various types of bacteria. Occasionally, in outbreaks of spontaneous pox in fowls as well as in flocks vaccinated with fowl-pox virus a marasmatic, no-lesion type of the disease is manifested. Beach[26] observes that, following vaccination "in some flocks of apparently healthy pullets and cockerels, there occurs rapid loss of condition and death of many birds for which no definite explanation is found." Similar results have been encountered by the authors in Kansas during the years 1928-30.

Infectious tracheo-bronchitis occurs as an acute, less commonly as a sub-acute and chronic, disease of the respiratory tract characterized pathologically by early hemorrhages of the trachea and the lungs,[27] accompanied by mild to severe inflammation of the trachea and bronchi. Clotting of the blood before or upon admixture with the mucus and inflammatory exudate results in the formation of amorphous masses or of casts which may extend throughout the respiratory tract. Hinshaw,[8] Beach[28] and Gibbs[12] refer to a chronic form of the disease, while Kernohan[6] observed that "a high percentage of birds recover spontaneously and rarely, if ever, does a chronic form develop." Graham[29] has recently described two distinct types of tracheo-bronchitis, a dyspneic and a toxemic type.

## CONTROL

Efforts to control virus-borne diseases of fowl have utilized, primarily, strict sanitary and police measures. Where these methods have failed and where eradication appears impractical or impossible, virus vaccination is frequently resorted to in recognition of the general character of virus diseases, namely, that recovery from infection in most instances effects an active immunity of significant duration.

Doyle[24] observed that, as a rule, if the first cases are killed early in an outbreak, the spread of fowl-pox may be easily circumscribed.

At present, the utilization of virus vaccination as a control measure against these diseases presupposes previous exposure of the flock, previous occurrence of the infection on the premises, or it anticipates subsequent exposure.

A variety of agents have been employed in attempts to stimulate immunity to fowl-pox artificially. Chief among these are fowl-pox virus (lesion material) from chickens and pigeons. The close relationship of the poxes is emphasized by the fact that chicken and pigeon viruses are immunologically indistinguishable although they differ in species adaptation. Advantage is taken of the latter situation in protective inoculation of the fowl against fowl-pox with pigeon virus.

It is now generally conceded that the use of highly attenuated or inactive fowl-pox virus is entirely unsatisfactory as a protective measure. Live-virus vaccination with fowl and pigeon viruses has recently been widely practiced in this country with favorable results.[19,21,30-37] As a consequence, a number of difficulties and factors which may greatly limit the application of fowl-pox virus vaccination have been brought to light. These include inoculation-pox (lesion and no-lesion forms) and failure to induce adequate or permanent immunity through attenuation and death of the inoculating material. The profound systemic effect induced in laying and in some healthy non-laying flocks, as well as in those with intercurrent diseases, has turned attention to the use of pigeon-pox virus. Encouraging reports by Doyle[24] and others in Europe have been followed by apparently satisfactory but limited trials with pigeon virus in the United States.[36,38]

The development of various methods of application of fowl-pox virus has been concerned with the factors of maximum time, efficiency in uniformly satisfactory vaccination, as indicated by a high percentage of inoculation point "takes" or reactions. The scarification method of de Blieck and van Heelsbergen[39,40] has been in this country largely superseded by the "follicle" and the "stick" methods of Johnson.[36] Introduction of the virus subcutaneously or by other routes is practically obsolete in view of the obvious epithelial affinity of the virus.

Studies to determine the period of time elapsing between inoculation or exposure and the development of demonstrable

immunity showed an average time of 20 to 31 days. Persistency of significant immunity has been demonstrated by Johnson,[38] 967 days after vaccination with fowl-pox virus.

That the use of fowl-pox virus produces a durable immunity to natural exposure is apparent from the field results reported by numerous investigators.

Attempts to stimulate immunity to infectious tracheo-bronchitis artificially have apparently been limited to the use of chemically attenuated or killed material (viscera and exudates)[6] and antisera.[11] As in the case of fowl-plague and similar diseases, satisfactory immunity was not induced by such means. Positive transmission experiments with filtrates of infective exudates and organs by intratracheal, intravenous, intramuscular, intraperitoneal or subcutaneous inoculation have been reported by Beach,[10] Graham,[11] Gibbs,[13] and Bushnell and Brandly,[12] while introduction of unfiltered exudates into the crop failed, in experiments by Beach[10] and Kernohan[6] to reproduce the disease.

## Experimental Studies

*Fowl-pox:* Inconsistent results in vaccination of field and experiment birds with the same strain of fowl-pox virus indicated a variable virulence in lesion material harvested at different periods after inoculation of birds used for virus-production. An experiment, involving two lots of virus-producing birds inoculated with the same strain and two series of nine test birds each, indicated that lesion material harvested the tenth and eleventh days after inoculation was the most virulent and produced the most extensive cutaneous lesions. Since a procedure of diluting virus-scab suspension was employed in making these determinations, a method of preparation of the lesion virus similar to that suggested by Kligler[41] was employed. It consisted of fine comminution of the dry pox scabs with sterile sand in a mortar and subsequent suspension in Locke's solution, saline or distilled water, followed by centrifugation for ten minutes at 1500 r.p.m. The supernatant fluid was assumed to contain virus bodies largely freed of the covering of protective protein. Preliminary tests revealed a much greater uniformity in dilutions than was the case with the sediment or with samples not subjected to centrifugation.

The desirability of standardizing the potency of pox virus was recognized by Beach[30] and his results indicate that for field

vaccination (subcutaneous inoculation) dilution of the entire scab suspension was satisfactory. Pyle[42] also attempted to determine a standard for virus vaccine for the cutaneous method. Our results indicate that closer determinations of potency may be made by employing the supernatant suspension than when the entire uncentrifuged scab suspension is used.

The possibility of pox lesion material carrying other poultry pathogens and thus artificially transmitting such diseases, was suggested in the light of similar results with this[35] and other virus vaccines. The use of chemical agents, including formalin, mercurochrome, phenol, brilliant green, crystal violet, acriflavine, chloroform, ether and glycerin, in quantities sufficient to destroy cultivable vegetative forms of bacteria, was found in all tests to alter markedly or inactivate entirely the pox-virus suspension employed. However, others have reported that glycerin does not injure the virus unless subjected to it for periods of approximately three to four weeks.

Attempts to infect pigeons with fowl-pox virus suspensions by heavy inoculation into fresh feather follicles and upon extensively scarified skin areas were negative in eight pigeons employed in the experiment. Subsequent inoculation of these eight individuals with pigeon-pox virus showed them apparently to be equally as susceptible as previously unvaccinated pigeons. The fowl-pox virus employed in the experiments was highly virulent, as indicated by the production of extensive lesions when inoculated into the combs of young White Leghorn cockerels. Mass inoculation upon the scarified combs of pox-susceptible fowls with pigeon virus resulted in only mild and transient comb lesions, with no perceptible systemic manifestations. However, on inoculation with fowl-pox virus after a period of one month, the pigeon-virus-treated fowls showed a perceptible although not absolute immunity. This immunity, referred to by Johnson[36] as a modified pox reaction, is characterized by the premature appearance of mild lesions without perceptible systemic reaction.

*Infectious tracheo-bronchitis:* Experimentation with this disease was confined largely to chicks as outlined in a previous paper.[12] The use of a number of strains of virus (3 from Kansas, 1 from Ohio, 1 from Illinois, and 2 from Kentucky) precluded the possibility that we were working with a local or atypical form of the disease. The close similarity between the symptoms and lesions of an epiornithic causing enormous losses in 1- to 4-week-old chicks during the 1930 hatching season, and infectious tracheo-

bronchitis in older birds, suggested an identical etiology. On subsequent trials with Berkefeld V and N filtrates of the tracheal exudates, lungs, livers and spleens of individual and composite samples from infected chicks, it was possible to reproduce with considerable uniformity the typical disease picture. Filtrates of tracheal exudate, of lungs and of liver and spleens of mature birds showing typical tracheo-bronchitis were capable, on instillation into the trachea of one- and two-week-old chicks, of reproducing an acute tracheo-bronchitis characteristic of that observed in the more rapid cases in natural outbreaks of the disease in chicks. In a number of cases it was proved that passage of strains originating in mature birds through chicks did not alter the infectivity of the filtrates for immature or mature birds. However, in no instance were strains of the virus originating in chicks shown to be capable of reproducing a typical infection in mature birds. Control chicks kept in the same building did not manifest spontaneous infection during the course of the experiment or during a subsequent three-week period.

A number of chickens of different breeds which had passed through artificially induced and spontaneous attacks of the disease at ages of one to five weeks were found, at the age of three to four months, to be susceptible to infection by large doses of filtrates of mature fowl strains. The mortality from tracheo-bronchitis in these birds was 75 per cent.

The substitution of yeast extract for physiological saline or distilled water as a vehicle for tracheal exudates and organ extracts was found to enhance Berkefeld filtration without apparent alteration of infectivity of the material. In none of the trials conducted was it shown that material passed through porcelain (Jenkins) filters reproduced the disease in susceptible birds.

Previous observations by us on the histopathology of the disease in chicks indicate the marked influence of simultaneous bacterial involvement. The significance of uncomplicated bacterial infection in inducing "gasping," a symptom considered pathognomonic of the disease in the field when supported by a satisfactory history, can also not be overlooked. This is particularly obvious in view of the wide prevalence of pullorum disease, and the fact that it may show an identical course.

These studies also revealed that involvement of the trachea and bronchi is usually more extensive than that of the larynx and hence the term, "infectious tracheo-bronchitis," is preferred over the designation, "infectious laryngo-tracheitis."

The macroscopic evidence of more severe and extensive hemorrhage in the lungs and trachea in cases of the infection in mature birds as compared with those in chicks was substantiated by microscopic study of these tissues from a number of mature fowl and from several hundred chicks.

## Discussion

In considering collectively some of the aspects of the virus diseases of fowls a striking similarity in certain features is obvious. Whether there is, among certain diseases now recognized as distinct entities, more than an apparent general or group relationship, magnified, at times, by the paucity of information regarding the etiology of the virus diseases, remains to be determined by further discriminating investigation.

The striking lack of information regarding the filtrable viruses perhaps accounts for attempts to differentiate somewhat similar diseases attributable to filtrable agents on the basis of symptomatology and distribution of lesions. Nevertheless, certain of the virus diseases of fowls manifesting a valid differential clinical picture and gross morbid anatomy have been found to be closely related immunologically. The work of Cooper,[43] indicating that Ranikheit disease in India, New Castle disease in England and avian or fowl-pest are immunologically identical, points to the comprehensiveness of the problem of differentiation and diagnosis. On the other hand, observations that the virus of fowl-pox was responsible for infectious bronchitis have been made. Gwatkin[44] later disproved his original observation on this point. Immunologically these diseases have not been shown to bear a significant relationship. Beach[28] reported that intratracheal instillations of fowl-pox virus produced lesions in some instances similar to those of fowl pox. van Heelsbergen[45] reported a disease from Holland similar to infectious bronchitis, which he ascribed to the virus of fowl-pox.

The appearance of diphtheritic lesions of the throat associated with a multiplicity of bacteria and other factors has presented a similar situation with regard to the identity of pox virus with the mucous-membrane form of fowl-pox. Reidmuller[46] has recently described a laryngitis and bronchitis with the formation of pseudomembranes. The colon bacillus was recovered from most of the cultures of this material. Filtrates of the pseudomembranes and exudates reproduced typical symptoms in ten days, and pox lesions also developed in the inoculated birds.

Unfiltered material required five days to effect the appearance of typical symptoms.

These and similar observations emphasize the difficulties arising in dealing with infections manifesting a disease picture indicative of pure or mixed virus infections complicated by an extensive and variable bacterial flora. Rivers[47] emphasizes that sufficient attention has not been accorded the effect viruses have upon each other when acting simultaneously or alternately in the same animal or to the effect that other kinds of diseases have upon the localization and activity of viruses.

The recent report of Kendall[48] on the artificial cultivation of certain filtrable agents, and the production of filtrable forms of common, known pathogens may foreshadow, with the development of knowledge concerning the filtrable viruses, a successful conquest of these important diseases.

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# STUDIES OF LEUKEMIA OF FOWLS*

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We began our studies of leukemia of fowls during October, 1928. Prior to that we noticed an increased number of birds received for routine diagnosis showed neoplasms of the liver, spleen, kidney and ovary.

## GROUP I

We purchased 17 White Minorcas from a flock of birds showing neoplastic changes in various internal organs. Eight of these birds died within one month after their arrival at our laboratory. The leucocyte blood-count of these eight birds varied from 8,400 to 27,110 per cubic millimeter of blood. All of these birds, when posted, presented enlargements of various organs. The nine remaining birds were kept under observation for one year and at that time were autopsied. None of this group presented neoplasms of the internal organs. The leucocytic blood-counts of this group varied from 16,000 to 48,000 per cmm. The birds with organ involvements failed to show leucocyte blood-pictures while under observation. Blood-smear examinations of this group of 17 birds showed no immaturity of any individual blood cells. Histological examinations of tissues from neoplastic organs presented infiltrations of lymphocytes similar to lymphatic leukemia.

Six apparently normal White Leghorns with leucocyte counts varying from 11,550 to 34,000 leucocytes per cmm. of blood, were placed in direct contact with the original group of birds. Seven apparently normal birds with leucocyte counts varying in a rather close range from 29,770 to 32,500 leucocytes per cmm. were injected intravenously in the wing vein with 2 cc of a filtrate obtained from a suspension of liver and spleen of a bird affected with leukemia. Six birds were given 10-cc intraperitoneal injections of a saline suspension made from liver and spleen obtained from the same source as the above lot. Blood counts and smear examinations were made monthly for a period of ten months. Our attempts to transmit this particular strain of lymphatic leukemia were unsuccessful.

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At the completion of the above experiment, we became interested in determining if there is a genetic basis for the occurrence of this disease, and if there is any greater susceptibility in one breed than in another. Nearly all the studies of this disease have been concerned with the histological pictures and blood conditions it presents in its various forms. Studies of this disease upon a genetic basis are beset with difficulties. If birds with clinical symptoms of leukemia were egg-producers, the problem would be very much simplified. Instead, however, it has been our experience that the ovary is very early involved in this disease and the hen either ceases egg-production or never begins.

We therefore obtained fowls from flocks in which leukemia had been diagnosed. From these birds, pedigreed chicks have been raised in the attempt to establish a strain of fowls susceptible to the disease. Up to the time of writing this paper, blood-cell counts, differential counts, and blood-smear examinations have been made monthly of all breeding birds in the experiment.

We started by obtaining a group of seven Plymouth Rocks, six pullets and one cockerel from Indiana. These birds originated from a flock where leukemia was known to be active. For identification purposes these birds were numbered 1938 to 1944, inclusive. We have had this group of birds under observation for slightly more than two years. Two hens have died (1941 and 1942). Hen 1941 presented lesions of mixed leukemia in the liver; other organs, normal. Hen 1942 presented no leucocytosis while under observation: the hemoglobin varied from 89 to 25 (Dare) just prior to death. Erythrocytes ranged from 3,000,000 to 1,150,000 just prior to death. Postmortem: liver, spleen and kidneys, atrophied. Histological examination of the liver presented evidence of intensive erythrocyte destruction, hemosiderosis. The lymphocytes of this bird showed budding of the large lymphocytes and azure granules in the cytoplasm of a few of the large lymphocytes. Granulocytes not involved. The four remaining hens (1938-39-40- and 43) have presented interesting blood smears: budding of the lymphocytes involving the medium and large-sized cells. If budding was well marked, we found a lymphocytosis. The differential counts showed from 58 to 90 per cent lymphocytes. The blood of one bird (1939) showed Auer bodies. These bodies lie in the cytoplasm of lymphocytes in a space resembling a vacuole and are rod-shaped and take the acid dye, in the Romanovsky stain. They very much resemble a tubercle bacillus in appearance. These bodies have

been described in cases of human lymphatic leukemia. The leucocyte count of these birds varied from 17,000 to 38,000, the highest count being 52,000. The hemoglobin varied from 45 to 99 (Dare). The erythrocytes varied from 1,740,000 to 3,600,000. The cockerel has a normal blood picture of : Hb, from 87 to 105 (Dare); leucocytes, 15,000 to 30,000, and erythrocytes, 2,500,000 to 5,500,000.

### Hatching Record and Results Obtained

*Bird 1938:* Seven chicks were hatched during 1930. Two died as baby chicks and later, one from laryngotracheitis. Two birds (1977 and 1966) died and had blood-pictures of lymphatic leukemia. Histological examination of tissues confirmed blood-picture. Eight chicks were hatched from 1938 during 1931. In all, 44 second-generation chicks were hatched during 1931.

*Bird 1939:* Seven chicks were hatched during 1930. Three died as young chicks. One died as result of roup; 1 cockerel and 2 hens living at present time. Cockerel 1961 has shown slight budding of lymphocytes, and 1969, during April, 1931, presented a good blood-picture of chronic mixed leukemia. This bird has not produced any eggs. Bird 1976, during February, 1931, had a blood-picture showing immaturity of lymphocytes, but the blood soon returned to normal. Bird 1968 showed mixed leukemia of blood and tissues. Bird 1974, lymphatic leukemia of blood and tissues. Thirteen chicks hatched from 1939 during 1931 and 28 second-generation chicks hatched during 1931.

*Bird 1940:* Four chicks were hatched during 1930. All died from other causes with the exception of one cockerel that has a normal blood-picture. Fifteen chicks have been hatched from this bird during 1931.

*Bird 1941:* This bird died, Nov. 1, 1930. The liver alone showed evidence of mixed leukemia. Four chicks were hatched during 1930, of which 3 are dead and 1 is living. The living bird (1992) has a good blood-picture of mixed leukemia with budding of lymphocytes well marked at the present time. Differential counts, 61 per cent lymphocytes and 38 per cent polymorphonuclear cells. Ten second-generation chicks have been hatched from this bird. A cockerel (1975) died in January, 1931, presenting gross lesions of lymphatic leukemia of the liver. Bird 1986 died June, 1931, and showed extensive budding of lymphocytes with vacuoles in the cytoplasm, a good blood-picture of lymphatic leukemia. The liver of this bird was slightly enlarged. Histological examination of this organ presented evidence of well-marked hemosiderosis and lymphocytic infiltration at the apex of the heart. Bird 1267 died April, 1931, and proved to be a very interesting case. In January, 1931, this bird had a leucocyte count of 21,550; February, 33,000; March, 44,880; two weeks later, in March, 21,000, and April, 124,000. The differential counts varied markedly: March, 85 per cent lymphocytes, 2 per cent polymorphonuclears and 10 per cent eosinophils; April, 28 per cent lymphocytes and 70 per cent eosinophils. No evidence of eosinophils in the tissues. The blood of this bird, when examined in April, presented a very good picture of mixed leukemia. Budding of lymphocytes was well marked, with vacuoles in the cytoplasm.

On account of the extensive enlargement of the liver, spleen, heart and kidneys, we inoculated 6 birds (1276, 7, 8, 9, 80 and 81,) on April 2, 1931, with Berkefeld N filtrates and suspensions obtained by making saline suspension from liver, spleen and kidneys. Birds 1276 and 1281 received 1 cc of the filtrate intravenously. These birds have been showing a gradual leucocytosis; blood smears show extensive budding of lymphocytes, while 1281 in addition has a blood-picture of mixed leukemia.

Birds 1278 and 1279 were injected intraperitoneally with 10 cc of the filtrate. Only a slight leucocytosis was evident one week after injection. Since then the leucocyte count has been practically within the normal range. A few lymphocytes with buds were found at the end of the first week, but the blood has been normal since.

Birds 1277 and 1280 were injected intraperitoneally with 10 cc of the saline suspension. The blood of 1277 has remained normal. The leucocyte count of 1280 has been within a normal range since injection. A few lymphocytes were showing buds one month after.

*Bird 1942:* This bird died, June 18, 1931. Blood smears showed only slight budding of lymphocytes. Other leucocytes normal. Anochromasia a few days prior to death: Hb, 25 (Dare); 1,150,000 erythrocytes. Postmortem: atrophied liver, spleen and kidneys. Histological examination of the liver showed pronounced hemosiderosis.

Only one chick (1982) was raised from this bird. It died in June, 1931. No second-generation chicks were obtained. Blood smears showed evidence of mixed leukemia with anochromasia. Extensive budding of lymphocytes with azure granules was present in the cytoplasm. The highest leucocyte count was 50,000. A short time prior to death, the differential count was 53 per cent lymphocytes, 40 per cent polymorphonuclears, 6 per cent eosinophils and 1 per cent mast cells. The internal organs were practically normal, with the exception of the ovary, which was enlarged but showed no evidence of leukemia. Four chicks were hatched from 1942 during 1931.

*Bird 1943:* Two chicks were hatched during 1930. One died of roup. The other bird was destroyed, April 2, 1931. The blood counts showed a gradual leucocytosis. Blood smears: slight budding of lymphocytes at beginning; later on, small lymphocytes were found with buds. Blood-picture: mixed leukemia. The internal organs were found normal upon gross examination with the exception of the kidneys, which were slightly enlarged. Histological examination of the liver, spleen, kidneys and ovary showed evidence of mixed leukemia. Twenty chicks were hatched from 1943 during 1931.

We also obtained 12 White Leghorn pullets from a flock where leukemia had been diagnosed, and the loss was quite severe (1919 to 1930, inclusive). Nine of the 12 died of different causes and, on postmortem examination, no evidence of leukemia was found. Blood smears of two of the remaining birds showed budding of the large and small lymphocytes for a short period of time. The liver of each of these birds showed a few areas of lymphatic leukemia. The remaining bird presented no evidence of leukemia.

We hatched 12 chicks from this group of birds, 9 of which died while quite young. One bird had normal blood-counts and cells, but the liver showed areas of mixed leukemia. The tissues of one bird were normal but there was budding of lymphocytes. The only living first-generation bird (1960) of this group has a varied leucocyte blood-count from 28,000 to 42,000, with extensive budding of lymphocytes and vacuoles in the cytoplasm of lymphocytes. Eight second-generation chicks have been hatched.

Another group of 13 Plymouth Rocks were obtained from a flock affected with leukemia. Four of these birds have died. Three of this number had an average leucocyte count of 32,880. Differential counts: 93 per cent lymphocytes, 5 per cent polymorphonuclears and 2 per cent eosinophils: Budding of lymphocytes was present. Postmortem: liver, very much enlarged; ovary, enlarged; other organs, normal. Diagnosis: lymphatic leukemia. No chicks raised from these three birds. The other

bird, just prior to death, had the following blood-picture: Hb (Dare), 43; leucocytes, 54,220; erythrocytes, 2,280,000. Differential: lymphocytes, 38 per cent; polymorphonuclears, 60 per cent and eosinophils, 2 per cent—a myelogenous leukemic blood-picture. Tissues showed no evidence of leukemia.

The nine living Plymouth Rocks of the group (1255-6-7-61-62-64-1995-97 and 99) have been under our observation for six months. All of these birds are showing different leucocyte counts, varying from 20,000 to 50,000. The individual cells show budding of lymphocytes and a few birds are showing a blood picture of mixed leukemia. The mixed-leukemia birds have as differential counts an average of about 52 per cent lymphocytes and 45 per cent polymorphonuclears. We have hatched 72 chicks from this group of birds with which to continue our studies.

TABLE I—*Average blood counts for 12 normal birds.*

| BIRD | HEMOGLOBIN (DARE) % | LEUCOCYTES | ERYTHROCYTES |
|---|---|---|---|
| 1952 | 78 | 27,610 | 2,410,000 |
| 1942 | 64 | 21,637 | 2,482,000 |
| 1279 | 73 | 24,727 | 2,762,500 |
| 1274 | 78 | 19,812 | 2,507,142 |
| 1961 | 83 | 24,125 | 2,698,571 |
| 1997 | 79 | 23,142 | 2,173,330 |
| 1964 | 80 | 23,282 | 2,765,550 |
| 1962 | 90 | 24,772 | 3,255,000 |
| 1257 | 73 | 29,292 | 2,822,850 |
| 1991 | 63 | 25,037 | 2,080,000 |
| 1992 | 75 | 25,130 | 2,298,550 |
| 1262 | 85 | 24,593 | 2,874,220 |
| Average | 76 | 24,425 | 2,594,188 |

Table I shows the average blood-counts and hemoglobin determinations for twelve birds made monthly for ten consecutive months.

## ERYTHRO-LEUCOSIS

We found one flock where the owner stated twenty-five or thirty birds had died, which, when opened, had enlarged and spotted livers. One bird (1264) was brought to the laboratory, February 17, 1931, for examination. The initial blood-count was as follows: 1,900,000 erythrocytes and 67,000 leucocytes. This bird was placed under observation and is still in our possession. Table II is a record of the blood-counts, differential counts and hemoglobin determinations:

TABLE II—*Record of hen 1264.*

| DATE (1931) | HB* (DARE) | LEUCOCYTES | ERYTHROCYTES | DIFFERENTIALS* | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | LARGE LYMPHOCYTES | SMALL LYMPHOCYTES | POLYMORPHONUCLEARS | EOSINOPHILS | MAST CELLS |
| 2–17 | 48 | 65,330 | 1,930,000 | | | | | |
| 2–26 | 50 | 84,220 | 2,150,000 | 20 | 16 | 1 | 63 | 0 |
| 3– 2 | 25 | 93,770 | 1,530,000 | 8 | 32 | 10 | 50 | 0 |
| 3– 3 | 20 | 96,220 | 1,580,000 | 30 | 16 | 48 | 6 | 0 |
| 3– 4 | 23 | 55,500 | 1,750,000 | 35 | 20 | 30 | 15 | 0 |
| 3– 5 | 37 | 49,770 | 1,500,000 | 19 | 30 | 46 | 5 | 0 |
| 3– 6 | 35 | 51,770 | 1,620,000 | 15 | 21 | 0 | 64 | 0 |
| 3– 7 | 26 | 13,110 | 740,000 | — | — | — | — | - |
| 3– 9 | 30 | 38,000 | 1,250,000 | 22 | 21 | 49 | 5 | 3 |
| 3–10 | 40 | 38,220 | 2,050,000 | 25 | 34 | 31 | 9 | 1 |
| 3–11 | 25 | 39,330 | 1,200,000 | 20 | 32 | 32 | 13 | 3 |
| 3–12 | 30 | 51,770 | 1,150,000 | 20 | 50 | 21 | 8 | 1 |
| 3–13 | 35 | 44,660 | 1,260,000 | 22 | 34 | 38 | 5 | 1 |
| 3–14 | 29 | 52,880 | 1,090,000 | 18 | 47 | 8 | 27 | 0 |
| | | | | LYMPHOCYTES | | | | |
| 3–21 | 55 | 44,220 | 1,720,000 | 53 | | 44 | 1 | 2 |
| 3–28 | 65 | 63,770 | 1,740,000 | 78 | | 17 | 3 | 2 |
| 4– 4 | 60 | 49,330 | 2,040,000 | 93 | | 5 | 2 | 0 |
| 4–11 | 50 | 45,770 | 2,300,000 | 84 | | 16 | 0 | 0 |
| 4–18 | 65 | 48,880 | 1,910,000 | 70 | | 30 | 0 | 0 |
| 4–25 | 60 | 25,880 | 1,540,000 | 81 | | 1 | 18 | 0 |

* Expressed in percentage.
— = not made.

This bird showed eosinophilic granules in the cytoplasm of the thrombocytes. On March 2, when smears were first studied for cytologic details, we found this bird's blood to contain many normoblasts, anochromasia, budding of lymphocytes, eosinophilic granules in the cytoplasm of thrombocytes and a well-marked eosinophilia. The flood of normoblasts continued for one week. Then the erythrocytes became nearly normal. The budding of lymphocytes continued for one week. Then the erythrocytes became nearly normal, but the budding of lymphocytes continued until April 25, at which time the blood picture had improved so that individual cells had become practically normal.

Another bird (1956) was obtained from a flock of White Leghorns where 25 per cent of the flock died from leukemia. We examined approximately 20 birds from this flock and histological and gross examinations established the diagnosis as lymphatic leukemia. We began blood studies of this bird, November 28, 1930, and continued until March 16, 1931, at which time the bird was destroyed. We attempted to transmit this strain of erythroleucosis by inoculation but failed. Table III shows the results of blood examinations.

TABLE III—*Results of blood examinations of bird 1956.*

| DATE | HB* (DARE) | LEUCOCYTES | ERYTHROCYTES | DIFFERENTIALS* | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | LARGE LYMPHOCYTES | SMALL LYMPHOCYTES | POLYMORPHONUCLEARS | EOSINOPHILS | MAST CELLS |
| 1930 | | | | | | | | |
| 11-28 | 55 | 66,866 | 3,690,000 | 32 | 37 | 16 | 15 | 0 |
| 12- 5 | — | 76,860 | 3,330,000 | — | — | — | — | – |
| 12- 9 | 62 | 51,770 | 3,290,000 | 25 | 45 | 0 | 30 | 0 |
| 12-29 | 82 | 34,000 | 2,770,000 | 30 | 60 | 6 | 1 | 3 |
| 1931 | | | | | | | | |
| 1-30 | 89 | 55,550 | 2,580,000 | 30 | 54 | 10 | 3 | 3 |
| 2- 6 | 85 | 62,220 | 3,630,000 | 15 | 69 | 15 | 0 | 1 |
| 2-19 | 65 | 57,220 | 3,010,000 | 63 | 18 | 18 | 1 | 0 |
| 2-26 | 59 | 57,550 | 2,010,000 | 18 | 56 | 22 | 2 | 2 |
| 3- 2 | 50 | 55,110 | 2,140,000 | 21 | 50 | 19 | 9 | 1 |
| 3- 9 | 49 | 57,110 | 1,850,000 | 15 | 50 | 20 | 15 | 0 |
| 3-13 | 49 | 50,000 | 2,220,000 | — | — | — | — | – |
| 3-16 | 43 | 55,110 | 1,880,000 | — | — | — | — | – |

* Expressed in percentage.
— = not made.

### CYTOLOGICAL DETAILS OF BLOOD-SMEAR EXAMINATIONS

11-28-30: Many normoblasts, vacuoles in cytoplasm of lymphocytes, eosinophilic granules in cytoplasm of thrombocytes, budding of lymphocytes and eosinophilia.

11-29-30: No counts made. More polymorphonuclears than on previous day; a slight improvement of blood.

12-5-30: Less budding of lymphocytes, but an increase in numbers of leukoblasts and older lymphocytes.

12-9-30: Budding of lymphocytes, immaturity of lymphocytes continued.

12-29-30: Budding of lymphocytes continued, erythrocytes nearly normal, more maturity in lymphocyte series.

1-30-31: Decrease in numbers of large lymphocytes, increase in numbers of small lymphocytes, budding of both types of lymphocytes, less immaturity of lymphocyte series.

2-6-31: Budding of lymphocytes continued. Numerous leukoblasts, increased immaturity of lymphocyte series.

2-19-31: Vacuoles in cytoplasm of large lymphocytes, eosinophilic granules in cytoplasm of thrombocytes, anochromasia, budding of lymphocytes. One cell was found that resembled a myeloblast.

2-26-31: Increased numbers of large lymphocytes with vacuoles in cytoplasm, numerous leukoblasts and younger cells of this type with nucleoli, and extensive budding of lymphocytes.

3-2-31: Slight improvement of individual blood-cells.

3-9-31: Extensive budding of large lymphocytes with very little cytoplasm. One cell found that resembled a reticulo-endothelial cell.

*Postmortem:* March 16, 1931. Liver enlarged about 1½ times, pale in color and very friable; spleen, normal; kidney, enlarged about 1½ times; ovary, practically ready to ovulate; intestines, no parasites; mesentery, thickened; heart, normal; lungs, normal; bone-marrow, very hyperplastic.

*Histological examination:* Bone-marrow, same type of cells as found in the peripheral circulation; lungs, normal; kidneys, lymphocytic infiltration surrounding glomeruli; heart, normal; intestines, normal; liver, areas of mixed leukemia surrounding hepatic blood-vessels. The bird was positive to pullorum test. Cultures taken at time of postmortem proved to be sterile.

## Discussion

Our efforts to transmit the strain of lymphatic leukemia that affected the group of White Minorca birds by injection of Berkefeld N filtrates obtained from liver and spleen of a bird affected with lymphatic leukemia were not successful. We injected the filtrate into the wing vein and into the peritoneal cavity, also made intraperitoneal injection of a saline suspension obtained from the same source as the filtrates. Contact birds failed to show evidence of the disease while under observation for ten months.

We received the group of Plymouth Rock birds from Indiana rather late in the season and were able to raise only 24 first-generation birds. Nine of these birds died as a result of coccidiosis, roup and laryngotracheitis. None of the tissues of this group showed any evidence of leukemia. Of the nine birds that died later on during the experiment, 2 birds showed lymphatic leukemia of the blood and tissues; one bird showed lymphatic leukemia of the blood; and another bird lymphatic leukemia of the tissues. Four birds showed mixed leukemia of the blood and tissues; and one bird showed only mixed leukemia of the tissues. Four of the six living birds of this group are showing evidence of lymphatic leukemia in blood smears. One bird has a mixed-leukemia blood, and the remaining bird, a cockerel, has normal blood-counts and blood-cells. We hatched this year 60 more first-generation chicks and 82 second-generation chicks. Not enough birds were hatched during the first year of this experiment to allow us to draw any conclusions as to the possibility of any hereditary factors being involved.

The blood study of the 6 birds injected, April 2, 1931, with filtrates and suspensions from bird 1267, a first-generation bird from our Indiana group, obtained from a hen whose liver alone showed evidence of mixed leukemia, shows that the birds injected with filtrate into the wing vein are showing a gradual leucocytosis and budding of lymphocytes, and the blood smears of one bird indicate mixed leukemia. Nothing of importance is being found in the four other birds injected with filtrate and suspension into the peritoneal cavity.

Our results with the group of 12 White Leghorn birds were not satisfactory. Two of the three birds that were raised showed evidence of leukemia. We have only one living first-generation bird from the group and her blood shows lymphatic leukemia at times, while there are periods when the blood-count and cells are

nearly normal. We have a few second-generation birds from this hen with which to continue our studies.

The second group of Plymouth Rock birds from present indications should be an interesting group to study. Their blood smears and counts show evidence of lymphatic and mixed leukemia.

A very interesting feature observed in our studies of leukemia of the fowl so far is a condition which we term an aleukemia, in other words, birds that show upon postmortem examination an enormous enlargement of the liver and spleen and in the majority of cases fail to show marked leucocytosis while under our observation.

We have mentioned Auer bodies having been found in the cytoplasm of large lymphocytes. We have had three birds showing these bodies; two of this group are still living, while the third bird, a cockerel, died. This bird showed a perivascular infiltration of lymphocytes in the liver. These Auer bodies are indicative of lymphatic leukemia in human cases.

Budding of lymphocytes has been quite regularly found in birds showing a gradual lymphocytosis. These buds are considered indicative of lymphatic leukemia.

The lymphocytes are classified with non-granular leucocytes, although some of them have non-specific azure granules of characteristic type. We found such granules in a few cases and have regarded them as an indication of a temporary functional condition of the cell. The number of such granules in the cytoplasm of these cells has varied. We have not observed these granules in the lymphocytes of normal blood of fowls and therefore their occurrence in pathological blood should be considered of importance and studied carefully.

We have also observed vacuoles in the cytoplasm of lymphocytes. These have been considered as functional alterations in the cells. They have not been observed by us in the blood of normal fowls. They are not an indication of immaturity.

We have also found eosinophilic granules in the cytoplasm of thrombocytes in some of the birds showing evidence of leukemic blood. They do not seem to be present constantly. No significance has been attached to their occurrence. No special study has been made of these cells, which are comparable to the platelets found in human blood. Their origin in human blood is well established, coming from the megakaryocytes, but very little is known regarding their origin in avian blood.


# TRICHOSTRONGYLUS COLUBRIFORMIS (= T. INSTABILIS) IN THE JACK RABBIT (LEPUS CALIFORNICUS MELANOTIS*)

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## Findings and Identification

December 1, 1928, the author autopsied a jack rabbit (*Lepus californicus melanotis*) which was killed in the cattle-yard on the University campus. Examination of the small intestine showed severe infestation with small nematodes. Specimens of these parasites were sent to the Zoological Division, U. S. Bureau of Animal Industry, Washington, D. C. The parasites were identified as *Trichostrongylus colubriformis* (= *T. instabilis*). The reply of December 8, 1928, stated also that "this nematode is a common parasite of ruminants, but has not been known to occur in jack rabbits or related mammals."

In February, 1929, five jack rabbits were killed in the vicinity of Lincoln, Nebraska, and autopsied. All were found to harbor large numbers of apparently these same parasites. Specimens from the small intestine of these rabbits were sent to Dr. Maurice C. Hall, Chief Zoological Division, Washington, D. C. Dr. Hall's reply of March 8, 1929, stated that the parasites were identified as *Trichostrongylus colubriformis.*

## Brief Review of Trichostrongylus

The *T. colubriformis* (1926) was found in sheep by (Gilles, 1892).

According to Brumpt,[1] these parasites are sometimes found in great numbers in the abomasum and duodenum of sheep and goats and may cause pernicious anemia. He states further that

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these parasites have been found by Looss, in 1895, in the intestinal contents of a number of cadavers. Also that they have been found in Japan by Ogata, of Tokyo, and by I. Ijima; in India by (Lane). Brumpt says:

> When these parasites are abundant in man, they are perhaps capable of producing pernicious anemia as found in animals.

Hegner, Root and Augustine,[2] in their book, "Animal Parasitology", say:

> Several species of Trichostrongylus are common and important parasites in the intestine of sheep and cattle, and rarely in man. *T. probolurus* (Railliet, 1896), *T. colubriformis* (Gilles, 1892) and *T. vitrinus* (Looss, 1905) occur in sheep and goats and have been reported in man. . . . . *T. extenuatus* (Railliet, 1898) is a common and widely distributed parasite of ruminants in the United States and is considered by some authors to be the cause of gastro-enteritis in calves. *T. capricola* (Ransom, 1907), occurs in goats and sheep of the United States and is apparently a more frequent parasite in these animals than *T. extenuatus*.

Marotel[3] gives a very good description of the pathogenic rôle of Trichostrongylus in the domestic animals.

Graybill[4] described the *T. affinis*, a new species in the wild rabbit. He writes:

> So far as the writer has been able to determine, three species of Trichostrongylus have been described from rabbits. *T. restortaeformis* (Zeder, 1800) Looss, 1905; *T. pigmentatus* (von Linstow, 1904) Hall, 1916; and *T. calcaratus* Ransom, 1911.

## Conclusions

Dr. Maurice C. Hall,[5] in referring to parasites in wild animals, said:

> Developments in the past indicate that we shall not have adequate knowledge for the protection of our live stock unless we take into consideration the parasites of wild animals. . . . Some of our most important parasites of domesticated animals come from wild animals on this continent. . . . Parasitism is not a static and fixed thing. Parasites are constantly adapting themselves to new hosts as opportunity offers.

The author thought perhaps it might be of interest to record the finding of *Trichostrongylus colubriformis* (= *T. instabilis*) in this new host, the jack rabbit.

## Acknowledgment

Credit is due Drs. Maurice C. Hall and E. W. Price, Zoological Division, Washington, D. C., for identification of these parasites.

### References

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[2]Hegner, R., Root, F. M., and Augustine, D. L.: Animal Parasitology. (New York and London, Century Co., 1929) p. 411.
[3]Marotel, G.: Parasitologie Vétérinaire. (Paris, J.-B. Baillière et Fils, 1927) pp. 145-157.
[4]Graybill, H. W.: A new species of round worm of the genus Trichostrongylus from the rabbit. No. 2548. Proc. U. S. Nat. Mus., lxvi (1929), pp. 1-3.
[5]Hall, M. C.: A parasitological résumé. Jour. A. V. M. A., lxxvii (1930), n. s. 30 (4), pp. 431-442.

## OSTEOGENIC CHONDROSARCOMA IN A DOG*

*By* E. L. STUBBS, *Philadelphia, Pa.*

*School of Veterinary Medicine, University of Pennsylvania*

*Subject:* The dog with this tumor was a 7½-year-old, brindle and white, female Boston terrier.

*History:* The owner on being questioned stated that one year previously the first disturbance was noticed, when the dog was less active and did not jump up on chairs as had been her custom. About four months previously, the right front limb began to enlarge gradually and was hard to the touch. About two months previously, it was brought to the Veterinary Hospital and at that time the tumor was approximately one-half its final size. One week preceding death, the appetite was very poor, and the dog was very weak and hardly able to walk. She was brought to the Veterinary Hospital to be destroyed. At this time the tumor mass in the region of the humerus caused a great enlargement of the entire front limb.

*Symptoms:* A blood-examination was made and showed red blood cells, 1,340,000; white blood cells, 19,200, of which polymorphonuclears were 81 per cent, small lymphocytes, 5 per cent and large lymphocytes, 4 per cent. The hemoglobin was 5 by the Sahli method. An x-ray was obtained (fig. 1) showing the shaft of the humerus totally destroyed and indicating that the tumor began in the shaft of the humerus. The x-ray shows the formation of small areas of bone tissue in the tumor. The head of the humerus is intact, as is the trochanter, but the shaft of the humerus is almost entirely destroyed by the extensive tumor growth.

*Autopsy:* Postmortem examination showed the right front limb enormously enlarged, with a tumor-like swelling the size of a man's head. It involved all the region of the right humerus extending from the neck to the carpal joint and looking almost as large as the dog itself. Several soft edematous areas were found on the neck, on the abdomen, near the foot and over the exterior of the tumor mass. The carcass weighed 22 pounds. The right front limb with the tumor weighed 8½ pounds, while the normal left front limb weighed 1⅛ pounds (fig. 2).

On being sliced the greater part of the tumor appeared gray and translucent (fig. 3). Several hemorrhages and several areas of cystic tissue were found mixed through it. A considerable

*Received for publication, December 18, 1931.

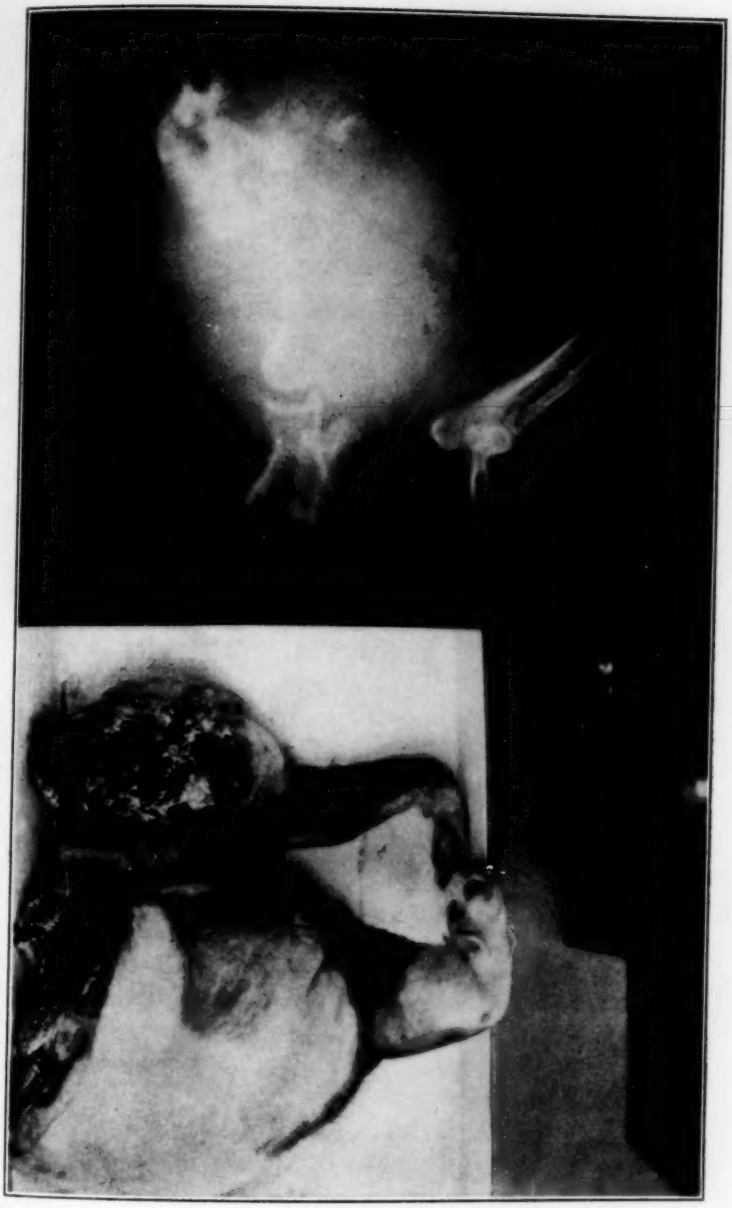

FIG. 1 (above). X-ray of osteogenic chondrosarcoma.
FIG. 2 (below). Two front limbs compared. Upper, normal; lower, tumor.

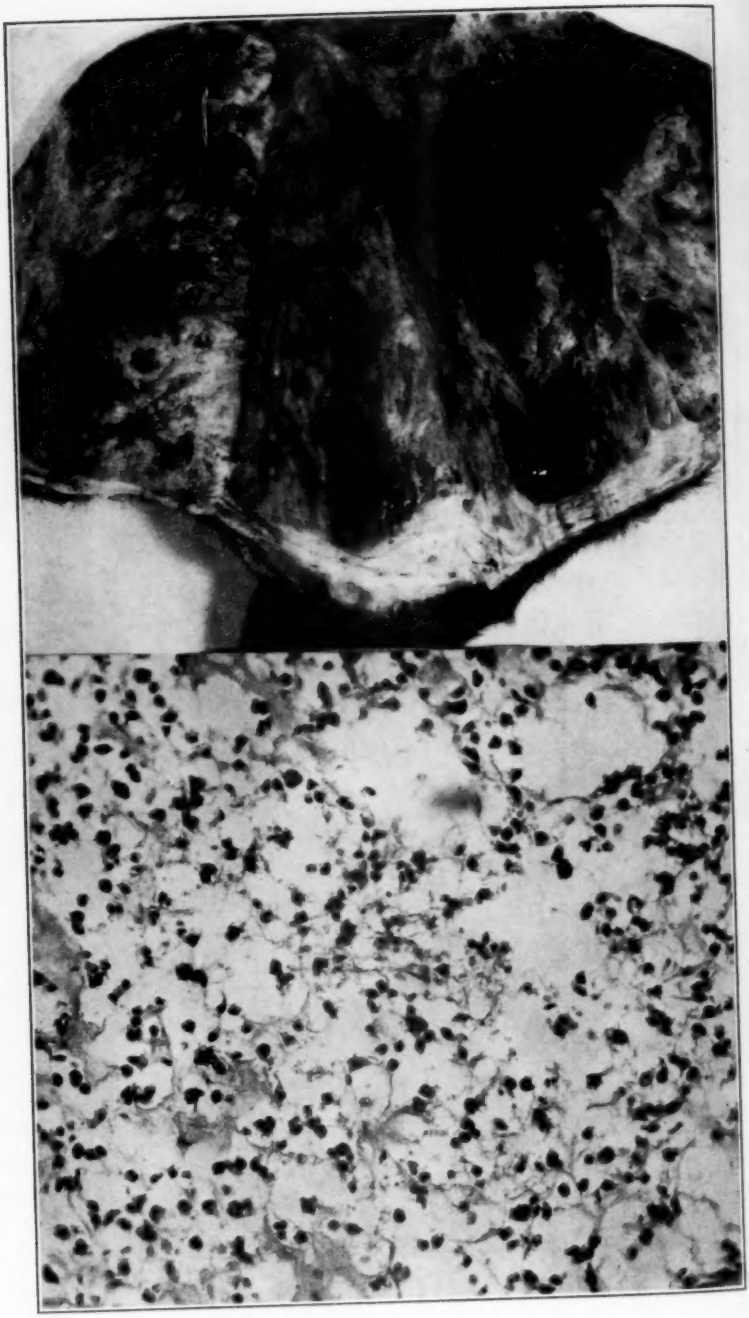

FIG. 3 (above). Sliced osteogenic chondrosarcoma.
FIG. 4 (below). Photomicrograph of osteogenic chondrosarcoma (x 170).

quantity of reddish, serous fluid oozed from the cut surface. The cut surface also had several areas with tissue resistant to the knife and a few areas of hard, bone-like tissue. The tumor was cut directly through where the humerus should have been, without any semblance of the shaft being found. The tumor did not seem to infiltrate the muscle of this leg but because of its great growth did push aside the muscle and caused atrophy of the muscle.

Postmortem examination showed no other alteration except in the lungs. The right middle lobe showed a tumor nodule in its edge and the right posterior lobe showed a tumor nodule in its center. Each nodule was about the size of a pea and was firm and gray on section.

*Diagnosis:* Microscopic examination (fig. 4) showed cellular areas and also areas with considerable stroma and some myxomatous degeneration. In many parts were cells of the cartilage type but without typical cartilaginous stroma. Some cells were round, others spindle cells. It is an osteogenic chondrosarcoma.

## THE FATE OF ANTHRAX BACILLI IN TICKS FROM AN ANTHRAX CARCASS*

*By* G. MARTINAGLIA, *Johannesburg, South Africa*
*Assistant Municipal Veterinarian*

On August 19, 1931, a cow in the Johannesburg quarantine live stock market died suddenly and, on examination of a blood smear taken from the carcass, anthrax was diagnosed.

The animal was heavily infested with ticks and a number of female blue ticks (*Boophilus decoloratus*) and female bont ticks (*Amblyomma hebraeum*) were collected to ascertain the presence and fate of anthrax bacilli in these ecto-parasites.

A bont tick and a blue tick (a) were destroyed and, from the alimentary tract of each, a smear was made and stained by Giemsa. On microscopic examination many anthrax bacilli were noted in the stained smears, the bacilli predominating in the one made from the blue tick.

Twelve hours after removal from the anthrax carcass, an engorged blue tick (b) was destroyed. Its dorsal cuticle was sterilized and then punctured by means of a sterile pipette, and ingesta were obtained from the alimentary tract. A few drops were streaked over a blood-agar slant. Thirty-six hours after

*Received for publication, March 11, 1932.

incubation, 42 anthrax colonies were observed. Twenty-four hours after removal from the carcass, a broth culture was made from the alimentary tract of an engorged blue tick (c). After incubation for 48 hours a typical fluffy-like anthrax growth was present.

A fourth engorged blue tick (d) was examined four days after removal from the anthrax carcass. Anthrax bacilli in the alimentary tract of the tick were still frequent but cultures made from it remained sterile.

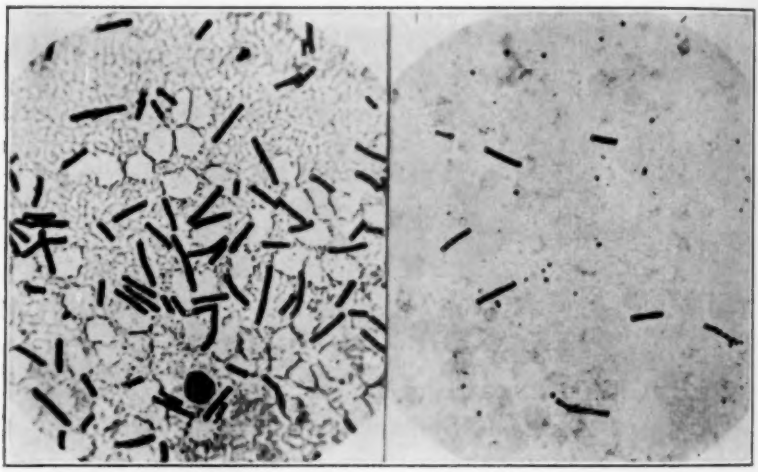

FIG. 1 (left). Anthrax bacilli in blood-smear of infected cow (x 600).
FIG. 2 (right). Anthrax bacilli in smear made from intestinal contents of blue tick (x 600).

The last living engorged tick (e) was destroyed on the eighth day after removal from its anthrax infected host. Anthrax bacilli were still present in the intestinal contents; the bacilli, however, stained more lightly and some appeared to be disintegrated. Cultures made from this tick also failed to grow.

From these observations it would appear that the anthrax organisms are still viable at least 24 hours after ingestion by the blue tick and then they are gradually rendered biologically sterile.

## RABIES QUARANTINE IN MICHIGAN

Due to the report of a definitely determined case of rabies at Lowell, Mich., quarantine has been ordered on 100 dogs in and around the town.


COMPLEMENT-BINDING PROPERTIES OF BRUCELLA ABORTUS OF BOVINE AND PORCINE ORIGIN. S. T. Walton. Jour. Immunol., xxii (1932), 1, p. 19.

Data show that the greatest variation in the complement-binding ability of antiporcine and antibovine sera, as measured by a number of bovine and porcine antigens, was 0.002 cc. At the peak of antibody production, and for a period of about twelve days before and a like period after the zenith was reached, there was a difference of only 0.0005 to 0.00008 cc. The amount of agglutinating antibodies present in a given sample of antiserum, whether it was antiporcine or antibovine, was apparently as great for one type of antigen as for the other. When there was a variation of antibody content in a sample, it was not constant for either type of antigen, but showed a difference for different strains of both types. In the absorption of agglutinins no antigen took appreciably more antibodies out of a serum for itself than it did for the other antigens and the small variation in the absorbing power was not constant for either type of antigen. The authors conclude that complement fixation as a means of detecting *B. abortus* antibody is on a par with the method of agglutination and suggest that the former might often be used as a confirmatory measure to detect a different type of antibody.

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THE INFLUENCE OF DIET ON THE DEVELOPMENT OF EXPERIMENTAL COCCIDIOSIS IN CHICKENS KEPT UNDER SANITARY CONDITIONS. Ena A. Allen. Amer. Jour. Hyg., xv (1932), 1, p. 163.

There is an indicated correlation between a high-protein and a high-vitamin diet and the production, among chickens infected with *Eimeria tenella*, of a form of coccidiosis which is subacute or chronic, with a relatively low daily production of oöcysts, a markedly flattened peak of oöcyst production on the second day after oöcysts appeared, a lessened amount of hemorrhage

which is also of shorter duration, a low mortality rate, and a failure to regain good physical condition by the eighteenth day after oöcysts appear, this latter presumably being associated with a continued oöcyst production in this more or less chronic form of coccidiosis. A low-protein and low-vitamin diet is correlated with a reverse of the above conditions. The presumption is that the high-protein or high-vitamin content of the diet is correlated with and is directly or indirectly responsible for the escape from severe acute coccidiosis and the production of a prolonged coccidiosis. Possibly both proteins and vitamins are correlated with the result. The mechanism of the result may be regarded as a form of resistance producing immunity to acute coccidiosis or resistance slowing the life cycle so that a predestined oöcyst production is slowed up and stretched over a long period of time instead of being rapidly completed with the production of acute clinical coccidiosis, or the resistance may involve both types.

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CUTANEOUS RETENTION OF INFECTIVE LARVAE OF THE DOG HOOKWORM ANCYLOSTOMA CANINUM, AND THE INFLAMMATORY REACTION TO SKIN PENETRATION. John E. Stumberg, Amer. Jour. Hyg., XV (1932), 1, p. 186.

Repeated skin infections on the same area of skin of white mice had no demonstrable effect upon the number of larvae which could be isolated from that area of skin 24 hours after the last infection. The same experiment repeated on dogs gave varying results. The conclusion is drawn that a local acquired immunity due to repeated skin infection has not been demonstrated. There seems to be some evidence that subcutaneous injections of extract of larvae caused a retention of larvae in the skin during penetration, but this is not definitely established. It is doubtful whether local passive immunization by subcutaneous injection of antihookworm sera had any effect upon the retention of larvae though some of the results considered by themselves might indicate such an effect. Two control animals given a skin injection without previous treatment showed results which indicated that the variability of the method itself was at fault. Histological study of the areas of skin showed that there was no relation between experimental procedure and degree of reaction. Attempted immunization had no effect on the face of intradermally injected dead larvae. The conclusion is reached

that the reaction to skin penetration was due to tissue injury by the larvae and not to an immune reaction to the larvae themselves.

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THE EFFECT OF RADIATION ON THE RESISTANCE OF CHICKENS TO FOWL CHOLERA. D. D. Donahue. Amer. Jour. Hyg., XV (1932), p. 206.

Over 500 chickens were exposed to various sources of radiation and then infected with *Pasteurella avicida*, using subcutaneous or intranasal inoculations. Daily exposures over a period of one month to a quartz mercury arc giving a total dose of 29 to 49 ZnS units, or a General Electric sun lamp (type S-I) giving a total dose of 63 to 189 ZnS units, slightly decreased the resistance of young chickens to subsequent subcutaneous injection with *P. avicida*. Daily exposure to a quartz mercury arc giving total doses of 54 to 203 ZnS units increased the resistance of young chicks to subsequent intranasal infection with *P. avicida*. Daily exposures to a General Electric sun lamp, giving total doses from 54 to 82 ZnS units, had no effect on the resistance of month-old chickens to subsequent intranasal infection with *P. avicida*. Chickens which were exposed to x-rays for 15 to 20 minutes and inoculated one hour after exposure were less resistant than the controls.

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A COMPARISON OF THE ANTHELMINTIC PROPERTIES OF HEXYLRESORCINOL AND HEPTYLRESORCINOL. P. D. Lamson, E. L. Caldwell. H. W. Brown and C. B. Ward. Amer. Jour. Hyg., XV (1932), 1, p. 306.

Heptylresorcinol has been shown to have approximately the same anthelmintic properties on ascaris *in vitro* as hexylresorcinol. Heptylresorcinol removed both ascaris and hookworms from dogs about as readily as hexylresorcinol. In two groups of cases treated under field conditions with hexylresorcinol and heptylresorcinol respectively, it was found that the hexylresorcinol removed a higher percentage of the hookworms, ascaris, and trichuris than heptylresorcinol.

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SOME CHEMICAL EFFECTS FROM CONSTANT INTRAVENOUS EPINEPHRINE INJECTION IN DOGS. Paul C. Samson and H. R. D. Jacobs. Amer. Jour. Physiol., XCIX (1932), 2, p. 433.

Epinephrine was administered intravenously to unanesthetized dogs at constant rates over relatively long periods of time (up to

13 days). Continuous epinephrine administration resulted in a transient hyperglycemia and glycosuria, both of which disappeared in 24 hours. The normal glycemia persisted with continued injection, even when the rate of administration was doubled, and there was no further glycosuria. The cessation of epinephrine administration, while the blood sugar values were within normal limits, led to an abrupt hypoglycemia with a slower return of blood sugar to normal values. With further injection a second hyperglycemia and glycosuria occurred, although not so marked as the first. Continuous epinephrine administration increased the urinary output in every case. Continuous epinephrine injection did not affect the urinary nitrogen excretion as measured by 24-hour periods.

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Infectious Oral Papillomatosis of Dogs. W. A. DeMonbreun and E. W. Goodpasture. Amer. Jour. Path., viii (1932), 1, p. 43.

The authors describe infectious papillomas occurring in the mouths of dogs. The virus of oral warts of dogs seems to possess greater cellular specificity than does the virus of human warts. Lesions could be induced only in the mouths of puppies although the skin and other mucous membranes were likewise injected with the virus. All healthy puppies appear to be susceptible. The incubation period is from 30 to 33 days in healthy puppies but may be as much as ten days longer in malnutritioned sickly puppies. Animals in which warts were definitely regressing could not be successfully reinoculated with the virus, although the regressing warts still contained active virus capable of infecting normal puppies. The limitation of the growth and initiation of the regression of the papillomas may be due to the development of immunity. Puppies that have recovered from the disease are immune to reinfection. Little is known regarding the proportion of older dogs that are susceptible.

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The Correlation Between the Histological Changes and the Fate of Living Tubercle Bacilli in the Organs of Tuberculous Rabbits. Max B. Lurie. Jour. Exp. Med., lv (1932), 1, p. 31.

The mononuclears of the liver, splenic pulp and bone-marrow destroy tubercle bacilli more rapidly than those of the lung, kidney or splenic corpuscle. The multiplication of tubercle

bacilli in an organ and their accumulation within mononuclears is accompanied by active new formation of these cells by mitosis. When these mononuclears are transformed into mature epithelioid cells and tubercles have reached their maximum development, the bacilli have already undergone extensive destruction and are disappearing. Tubercle bacilli of moderate virulence are usually effectively destroyed within epithelioid cells of all organs. In the lungs and kidneys bovine bacilli persist within epithelioid cells, but in other organs they are usually destroyed. Tubercle bacilli are less effectively destroyed within epithelioid cells collected in the alveoli of the lungs than in those forming tubercles in the interstitial tissues. After multiplication of tubercle bacilli has ceased, regeneration of mononuclears by mitosis becomes less active and now giant-cells may be formed from preëxisting epithelioid cells. Lymphocytes and encapsulation of tubercles by granulation tissue do not cause the destruction of tubercle bacilli. Immediately after infection, accumulation of the less virulent types of tubercle bacilli in the tissues does not cause caseation and the more virulent bovine bacillus produces this change only in the lung. Later caseation occurs in the presence of a small number of bacilli, and must be thought of as due, in part at least, to sensitization.

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THE EPIDEMIOLOGY OF FOWL CHOLERA. VI. The spread of epidemic and endemic strains of *Pasteurella avicida* in laboratory populations of the normal fowl. I. W. Pritchett and T. B. Hughes. Jour. Exp. Med., lv (1932), 1, p. 71.

Strains of *Pasteurella avicida* from "spontaneous epidemics" of fowl cholera, when introduced intranasally in fixed doses into specially bred chickens, induced fatal fowl cholera in about 40 per cent but did not survive in the nasal clefts of resistant birds nor spread to normal contacts. The failure of epidemic strains of *P. avicida* to survive in nature and to spread among specially bred chickens in the experiments is not understood. The authors suggest the possibility that they require hosts of abnormally low resistance or the presence of some other agent which as yet has not been discovered. Strains of *P. avicida* from "spontaneous endemics" of fowl cholera, when introduced similarly into chickens, failed to kill but did survive in the nasal clefts of inoculated birds and spread rapidly to normal contacts. "Laboratory" variants of the epidemic strains of *P. avicida* failed to kill or survive in the test birds and did not spread to contacts.

THE ACCUMULATION OF IRON IN TUBERCULOUS AREAS. II. Survival time of tuberculous rabbits injected with ferric chloride. Valy Menkin. Jour. Exp. Med., lv (1932), 1, p. 101.

Repeated intravenous injections of ferric chloride are followed by an increase in the survival time of tuberculous rabbits. In the particular series of experiments reported this increase amounts to about 78 per cent over the average survival time of control rabbits. Tuberculous animals repeatedly injected with ferric chloride increase in weight during part of the period of these injections. The level reached in the series studied markedly exceeds that attained by control rabbits. Both control and experiment animals die of generalized tuberculosis. There is no indication at the time of death of any differences in the degree of pathological involvement between the two groups of animals.

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THE INFLUENCE OF AGE AND OF DURATION OF TREATMENT ON THE PRODUCTION AND REPAIR OF BONE LESIONS IN EXPERIMENTAL HYPERPARATHYROIDISM. Henry L. Jaffe, Aaron Bodansky and John E. Blair. Jour. Exp. Med., lv (1932), 1, p. 139.

Young guinea pigs are more susceptible than adult guinea pigs to the effects of single or repeated doses of parathormone, as shown by the bone changes. Several successive daily doses of parathormone, in rapidly increasing amounts, result in an accentuation of the effects. In young and adult guinea pigs a compensation is established during prolonged parathormone treatment, which permits considerable repair of bone lesions produced earlier in the treatment.

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## Arizona and New Mexico Veterinarians Plan Joint Meeting

At an informal meeting of the members of the Arizona Veterinary Medical Association, held at the office of Dr. J. C. McGrath, in Phoenix, April 12, 1932, it was decided to accept the proposal of the New Mexico Veterinary Medical Association to hold a joint meeting at Springerville, Ariz., June 12, 1932. The New Mexico veterinarians have given assurance of a large attendance and as Springreville is situated at the head of the Coronado Trail, in the heart of the "Switzerland of America," it should draw the entire Arizona membership at that season of the year.


## Regular Army

Colonel Wm. P. Hill is relieved from further assignment and duty at the Presidio of San Francisco, Calif., effective in time to sail on transport scheduled to leave San Francisco, Calif., on or about April 4, 1932, for New York, and upon arrival will proceed to Fort George G. Meade, Md., for duty as station veterinarian and additional duty as attending veterinarian, Fort Howard, Md.

Major Oness H. Dixon, Jr. is relieved from duty at Fort Jay, N. Y., effective in time to sail from New York City on or about June 18, 1932, for the Hawaiian Department.

First Lieutenant Stanley McL. Nevin is relieved from his present assignment and duty at Fort Riley, Kans., effective August 25, 1932, and will then report to the commandant, the Cavalry School, Fort Riley, for duty as a student in the 1932-1933 troop officers' course.

## Veterinary Reserve Corps

*New Acceptances*

Niemann, Karl Wm.....2nd Lt...721 Mill St., Reno, Nev.

*Promotions*

| | To | |
|---|---|---|
| Doerr, John | Major... | Melbourne, Ia. |
| Marsh, Edward Thornton | Capt.... | Globe Lab., Ft. Worth, Texas |

*Changes of Address*

| | | |
|---|---|---|
| Bailey, Wm. Westly | 2nd Lt... | B. A. I. State House, Trenton, N. J. |
| Barrett, John Henry | 2nd Lt... | 1 Newton Ave., Westerly, R. I. |
| Cunningham, George Nelson | Capt.... | 8227 Vernon Ave., Chicago, Ill. |
| Douglass, Oliver Tyler | 2nd Lt... | Pet Animal Hosp., Davenport, Ia. |
| Dwyre, Raymond R. | 2nd Lt... | 920 East Chamberlin St., Dixon, Ill. |
| Fletcher, Pearl Clifford | 2nd Lt... | 216 S. Center St., Clinton, Ill. |
| German, Walter Allen | 2nd Lt... | 6527 S. Stewart Ave., Chicago, Ill. |
| Keck, Wm. Carl | Capt.... | 2150 Union Blvd., Grand Rapids, Mich. |
| Lavender, James Blaine | 2nd Lt... | 1840 S. 27th St., Lincoln, Nebr. |
| Marzillier, Otto Reinhold | 2nd Lt... | 721 Church St., Jefferson, Wis. |
| Price, Clayton John | 2nd Lt... | 624 E. Mansur Ave., Guthrie, Okla. |
| Rath, Reuben Benjamin | Capt.... | 134 W. Van Buren St., Battle Creek, Mich. |
| Rinehart, Harvey Monroe | 2nd Lt... | Broad St., Knoxville, Ill. |
| Stokes, Sidney Walter | 1st Lt... | 318 N. Central Ave., Chicago, Ill. |

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## Golden Wedding Anniversary

Dr. and Mrs. George C. Faville, of Richmond, Virginia, will celebrate the fiftieth anniversary of their marriage. May 6, 1932. Congratulations!


## VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held Wednesday, February 3, 1932, in the Academy of Medicine Building, 103rd St. and Fifth Ave., at 8:30 p.m.

Dr. O. E. McKim, President, called the meeting to order and introduced Dr. G. A. Dick, Dean of the Veterinary School of the University of Pennsylvania, who spoke very ably on "Heredity and Its Workings." Dr. Dick brought out the fact that while disease is not inherited, structural weaknesses are. He mentioned examples such as club feet, hernia, tumors, hemophilia and color-blindness (which is handed down by the female). Dr. Dick used lantern-slides to illustrate his talk. The members of the Association extended a rising vote of thanks to him at the conclusion of the lecture.

Dr. R. S. MacKellar, Sr., reported a case of fracture of the os suffraginis in a polo pony, with a subsequent good recovery.

Dr. R. S. MacKellar, Jr., gave a report for the Banquet Committee. Dr. McKim appointed the committees to serve for the coming year.

JOHN E. CRAWFORD, *Secretary*

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## FIFTH ANNUAL CONFERENCE OF WORKERS IN PULLORUM DISEASE CONTROL

The fifth annual Conference of Workers in Pullorum Disease Control was held at Cornell University, Ithaca, N. Y., April 4-6, 1932. Twenty-five members were in attendance from Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Ohio, Ontario, Pennsylvania, Vermont, Virginia, West Virginia and the Bureau of Animal Industry, U. S. Department of Agriculture.

The program consisted of reports on the activities and accomplishments of the Antigen Committee; data concerning con-

trol methods and testing results submitted by the various states and compiled by Dr. E. L. Brunett, chairman of the Conference; discussions concerning studies of the growth of *S. pullorum* on various culture media and the comparative agglutinability of the resultant antigens; antigen studies; standard methods of diagnosis; comparative results of the whole-blood agglutination test and the tube agglutination test; prevention of "jelled" samples; problems involved in the preparation and use of the McFarland nephelometer; short-interval testing in the eradication of pullorum disease; transmission of pullorum disease among sexually immature pullets; detection of agglutinins in chicks; exposure of birds to soil and litter contaminated with feces from infected birds; avenues of infection; infecting females by feeding fresh eggs laid by infected hens; susceptibility of guinea fowl, pigeons and sparrows to *S. pullorum* infection; rules and regulations for accreditation; quarantine and disposal of reactors with proper follow-up measures; decrease in egg-production associated with blood-collection; and demonstration of the whole-blood agglutination test.

The following poultry diseases other than pullorum disease were considered: Leucosis; tumors; fowl-pox immunization (methods and viruses employed); infectious laryngotracheitis and coccidiosis.

Correspondence relative to the 1933 Conference should be addressed to Dr. C. L. Martin, University of New Hampshire, Durham, New Hampshire, chairman of the committee on selection of place and time for the next conference.

*Summary of 1931-32 testing data submitted by the laboratories.*

| Laboratory | Samples Tested | Positive (%) | Flocks | Positive |
|---|---|---|---|---|
| Connecticut | 189,498 | 1.4 | 219 | 66 |
| Cornell | 73,166* | 4.7 | 138 | 120 |
| Delaware | 97,157 | 3.6 | 309 | 276 |
| Farmingdale | 7,899* | 0.8 | 16 | 11 |
| Maine | 133,071 | 0.4 | 237 | 27 |
| Maryland | 26,518 | 13.3 | 71 | 71 |
| Massachusetts | 420,861 | 0.9 | 455 | 100 |
| New Hampshire | 205,252 | 0.5 | 196 | 27 |
| North Carolina | 104,937 | 3.5 | 421 | 307 |
| Ontario | 162,937 | 3.4 | 557 | 395 |
| Pennsylvania | 309,880 | 5.2 | 620 | 544 |
| Rhode Island | 7,531 | 2.8 | 15 | 11 |
| Vermont | 38,425 | 2.7 | 88 | 25 |
| Virginia | 126,168 | 8.9 | 635 | 529 |
| West Virginia | 34,032 | 8.7 | 231 | 193 |

*Represents number of birds tested.

## ILLMO VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Illmo Veterinary Medical Association took place at Collinsville, Ill., April 8, 1932. The Saint Louis District Veterinary Medical Association met at the same time and place, instead of holding the regular April meeting.

About forty veterinarians were present, and the meeting was most enthusiastic. The forenoon was devoted to a clinic, at which a number of very interesting cases were presented for diagnosis and operation. There was a literary program in the afternoon, and several pertinent subjects were discussed. All officers were re-elected for 1932: President, Dr. H. J. Schlesinger, New Athens, Ill.; vice-president, Dr. L. J. Miller, Waterloo, Ill.; treasurer, Dr. Wm. Beckman, Saint Louis, Mo.; secretary, Dr. H. R. Schwarze, East Saint Louis, Ill.

H. R. SCHWARZE, *Secretary*

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## NORTHWESTERN PENNSYLVANIA VETERINARY MEDICAL CLUB

The regular quarterly meeting of the Northwestern Pennsylvania Veterinary Medical Club was held April 12, 1932, at Erie. Sixteen members attended. Dinner was served at the Hotel Fischer, followed by a literary program at the Chamber of Commerce rooms.

A very interesting and instructive talk was given by Dr. B. S. Putts, a prominent Erie physician, on the subject, "The X-Ray in Veterinary Practice." Dr. Putts used several films to illustrate his talk, showing fractures, dislocations, and foreign objects such as are frequently swallowed by animals. The x-ray is valuable in establishing an accurate diagnosis in this type of case and should be used by all practicing veterinarians.

Dr. C. J. Marshall, of the School of Veterinary Medicine, University of Pennsylvania, lectured on "The Nervous Conditions and Diseases of Animals."

Dr. John Bryce, of Erie, was present at the meeting. He is the oldest living graduate of the Ontario Veterinary College, having been graduated with the class of 1870.

Dr. E. E. Bittles, of Waterford, and Dr. W. C. Wootton, B. A. I. inspector, stationed for the past two years at Schaffner Brothers packing-plant, met for the first time since their graduation from the Ontario Veterinary College in 1890.

It was voted to hold the July meeting at the certified milk farm of Dr. Frederick Taylor, at Pulaski, Lawrence County.

P. L. ROUSE, *Secretary*

## EAST TENNESSEE VETERINARY MEDICAL SOCIETY

The East Tennessee Veterinary Medical Society met March 12, 1932, at Morristown, Tenn., with Drs. W. B. Lincoln, G. P. Whittington and R. E. Baker as joint hosts. Dr. J. F. Kagey, of Kingsport, presided.

The program was featured by a dinner at Cherry Villa, the home of the secretary. Dinner was followed by a very interesting literary program at the Terminal Hotel, the main feature of which was a well-prepared lecture by Dr. W. B. Lincoln, entitled "Postmortem Diagnosis of Tuberculosis in Hogs and Cattle." A thorough and general discussion of no-lesion and skin-lesion reactors followed the lecture. Much evidence was brought to light to support the belief, entertained by many, that skin tuberculosis in cattle originates from the human type of the tubercle bacillus.

The Society went on record as favoring the appropriation of $20.00 for the benefit of the A. V. M. A. meeting at Atlanta. All members of this Society have made, through the Tennessee Veterinary Medical Association, of which they are all members, a substantial appropriation. This is a double proof of their loyalty.

R. E. BAKER, *Secretary*

## ROCK COUNTY VETERINARY MEDICAL ASSOCIATION

At the annual meeting of the Rock County Veterinary Medical Association, held in Janesville, Wis., April 13, 1932, the following officers were elected for the ensuing year: President, Dr. R. L. Brown, Janesville; vice-president, Dr. Claire G. Culham, Stoughton; secretary-treasurer, Dr. A. N. Lawton, Brodhead. The meeting was held at the home of Dr. L. J. Lewis, who gave a talk on "Diseases of Poultry."

## STATE BOARD EXAMINATION

Nebraska Bureau of Examining Boards. State House, Lincoln Nebr. June 24-25, 1932. Application must be on file at Bureau, 15 days prior to examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Nebr.

# PERSONALS

## MARRIAGES

Dr. Erwin L. Jungherr (Vienna '22), of Storrs, Conn., to Miss Marie Frances Healy, of New Haven, Conn., March 28, 1932, at New Haven, Conn.

Dr. T. J. Leasure (K. S. C. '30), of Lawrence, Kans., to Miss Eugenia Lauck, of Maple Hill, Kans., February 13, 1932, at Maple Hill, Kans.

Dr. C. C. Nickel (Chi. '20), of Nowata, Okla., to Miss Thelma Demory, of Tulsa, Okla., April 8, 1932, at Tulsa, Okla.

## BIRTHS

To Dr. and Mrs. E. A. Schmoker, of Evanston, Ill., a daughter, Juanita Elaine, February 2, 1932.

To Dr. and Mrs. E. R. Frank, of Manhattan, Kans., a son, Robert, February 23, 1932.

To Dr. and Mrs. Leland C. Lynch, of Middletown, Ohio, a son, Leland Jr., March 16, 1932.

## PERSONALS

Dr. L. F. Vaughn (K. C. V. C. '16) has removed from Gilroy, Calif., to San Jose, Calif.

Dr. C. C. Dauber (Ont. '04), of Sturgis, Mich., is municipal meat inspector for his city.

Dr. G. W. Worrell (St. Jos. '17) has changed location from Grenada, Calif., to Redding, Calif.

Dr. V. M. Kaliher (McK. '18), of Henry, Ill., has accepted a position in the U. S. Bureau of Animal Industry.

Dr. H. Alme (Ont. '31) has opened the South Shore Veterinary Hospital, at 5466 Lake Park Ave., Chicago, Ill.

Dr. Leonard R. Richardson (O. S. U. '31), formerly of Akron, Ohio, is now at 71 W. Frambes Avenue, Columbus , Ohio.

Dr. O. F. Butterfield (Chi. '96), who has been in practice at Tiskilwa, Ill., since last September, has removed to Henry, Ill.

Dr. C. D. Bashore (Cin. '15), of Shelby, Ohio, has been appointed a field veterinarian in the Ohio Department of Agriculture.

Dr. Harry J. McCauley (Chi. '18), who was in practice at Tripp, S. Dak., has returned to his former location at Beresford, S. Dak.

Dr. Leonard P. Bailey (O. S. U. '31), formerly of South Charleston, Ohio, reports a new address: 509 S. Main St., Piqua, Ohio.

Dr. Otto Stader (U. P. '18) is contemplating the erection of a veterinary hospital at Geneva, Ill., at an estimated cost of $10,000.

Dr. C. A. McKillip (McK. '09), formerly of Highland Park, Illinois, is now at Saint Charles, Illinois. Address: 715 W. Indiana St.

Dr. P. L. Ellis (Iowa '13), who took postgraduate work at Ohio State University during the past year, has returned to Merrill, Iowa.

Dr. J. W. Childs (Colo. '28), who has been with Dr. J. E. Van Sant, of Bakersfield, Calif., is now located at Verdugo City, Calif.

Dr. R. D. Macintosh (Ont.'11), who has been with the Tela Railroad Company in Honduras for several years, has returned to Scotland.

Dr. A. L. Birch (Iowa '21), who has been Deputy Live Stock Inspector, County of Los Angeles, Calif., has gone to Worthington, Minn.

Dr. Chester L. Nelson (Ont. '11) has removed from Kiowa, Colo., to Clarence, Mo., where he has taken over the practice of Dr. W. D. Howe.

Dr. W. D. Howe (Chi. '16), formerly of Clarence, Mo., has moved to Hannibal, Mo., where he has established the Junction Dog and Cat Hospital.

Dr. O. H. Geib (Gr. R. '14) was elected mayor of Corunna, Mich., on April 4. He received 463 votes to 413 for his opponent, who has held the office for three terms.

Dr. S. C. Lilly (O. S. U. '16), until recently employed by the city of Dayton, Ohio, as meat and milk inspector, has located at London, Ohio, for general practice.

Dr. H. R. Holmes (Ind. '15), of Lyons, Ind., received an appointment in the U. S. Bureau of Animal Industry and has been assigned to meat inspection in Chicago.

Dr. Lewis Spolum (Iowa '03), who was in partnership with his brother at Watertown, S. Dak., has located at Olivia, Minn., where he will practice on his own account.

Dr. Hamlet Moore (Ont. '98) reports that he is moving his office and hospital from 622 N. Rampart St., to larger quarters at 2941 Grande Route St. John, New Orleans, La.

Dr. T. E. Palmer (Ind. '10), of Casey, Ill., recently declined an appointment in the U. S. Bureau of Animal Industry at Albert Lea, Minn., and has decided to remain in practice.

Dr. J. L. Tyler (Chi. '89) reports a change of address from Pomona, Calif., to Oroville, Calif., made necessary on account of his work as meat inspector for the State of California.

Dr. Andy Crawford (K. S. C. '30), of McComb, Miss., has received an appointment in the U. S. Bureau of Animal Industry and is stationed at Newark, N. J., on meat inspection.

Dr. A. Slade (Cin. '13), of Falmouth, Ky., has sufficiently recovered from the effects of his recent automobile accident to be able to look after his practice in Grant and Pendleton counties.

Dr. Morton Hattery (McK. '07) was retired from the service of the U. S. Bureau of Animal Industry, March 31, 1392. He has changed his residence rom Chicago, Ill., to Dayton, Ohio.

Dr. C. E. O'Neal (K. S. C. '16) is state veterinarian of Mississippi, succeeding Dr. G. B. Bradshaw (A. P. I. '19). Dr. O'Neal was formerly instructor in veterinary science at the Mississippi State College.

Dr. E. S. Norton (U. P. '11), of Greenville, Miss., has assumed the duties of meat and milk inspector, in coöperation with the U. S. Public Health Service and the Mississippi State Board of Health, in Meridian, Miss.

Dr. F. J. Emmer (Ont. '08-'16), of Richmond, Mich., has returned to Shepherd, Mich., for general practice, after an absence of fifteen years. Part of this time he was a veterinary inspector for the Detroit Board of Health.

Dr. Robt. S. MacKellar (N. Y. C. V. S. '94), of New York, N. Y., took a trip to Bermuda, during the month of February. He was accompanied by Mrs. MacKellar. This was the first real vacation for Dr. MacKellar in almost twenty years.

Dr. E. R. Braun (Wash. '29), formerly of Sacramento, Calif., has been transferred by the State Department of Agriculture, and is doing tuberculosis control area work in Los Angeles County. He is now at 6913 Middleton St., Huntington Park, Calif.

Dr. C. C. Hisel (K. C. V. C. '16), state veterinarian of Oklahoma, has been appointed a member of a committee to draft a bill that will provide for statewide postmortem meat inspection, to be presented at the next session of the Oklahoma legislature.

Dr. John T. E. Dinwoodie (U. P. '13), who has been managing editor of the *Dakota Farmer*, at Aberdeen, S. Dak., for a number of years, has resigned to accept a position in the Extension Department of the North Dakota Agricultural College, at Fargo.

Dr. V. H. Miller (O. S. U. '24), of Ottawa, Ohio, has started suit against the Mid-West Motor Freight Company, of Detroit, Mich., in the sum of $10,000 for personal injuries and $2,239.85 for medical attention and hospital service. Dr. Miller was injured by a truck owned by the defendant and parked illegally on the Dixie Highway.

Dr. H. F. Wilkins (K. C. V. C. '16), Chief Deputy State Veterinarian of Montana, recently returned to Helena, after a very pleasant vacation. Accompanied by Mrs. Wilkins, he left Helena the middle of December. He spent about three weeks in Mexico and a month in California, visiting his brothers and a number of other veterinarians in the Golden State.

## BUREAU TRANSFERS

Dr. James M. Wingate (A. P. I. '28) from Newark, N. J., to Birmingham, Ala., on tuberculosis eradication.

Dr. Guy T. Cole (U. P. '02) from New Orleans, La., to Jacksonville, Fla., on meat inspection.

Dr. G. B. Bradshaw (A. P. I. '19) from Birmingham, Ala., to Nashville, Tenn., on tuberculosis eradication.

Dr. J. H. Sharp (K. C. V. C. '12) from Denver, Colo., to Albuquerque, New Mexico, on field inspection.

Dr. Alpha E. Wardlow (O. S. U. '17) from Sacramento, Calif., to Lincoln, Nebr., on field inspection.

Dr. James E. Ewers (K. C. V. C. '10) from Albuquerque, N. M., to Denver, Colo., on meat inspection.

Dr. S. K. Nelson (McK. '17) from Albert Lea., Minn., to Philadelphia, Pa., on meat inspection.